



*SECOND EDITION*  
CLINICAL ROENTGENOLOGY  
OF THE  
CARDIOVASCULAR SYSTEM

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ROESLER



SECOND EDITION, SECOND PRINTING

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# Clinical Roentgenology *of the* Cardiovascular System

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BY

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*IN GRATITUDE*

to

H. FLANDERS DUNBAR, M.D., PH.D , D.MED SC.

W. EDWARD CHAMBERLAIN, B.S., M.D.

Who have given me Vision and Courage  
for this Task

Go forth my painefull booke,  
Thou art no longer mine:  
Eache man may on thee looke,  
The shame or praise is thine.

What delight with me remaine

Go forth in Gods name.

And seeke thou for no praise,  
No thanke nor yet reward,  
Nor eache man for to please,  
Haue thou no great regard.

For as to pleasure many,  
I haue bene euer glad,  
Right so to displease any,  
I would be loth and sad.

The labour hath bene myne,  
The trauaile and the paine,  
Reproches shall be thine,  
To beare we must be faine

But if thou please the best,  
And such as be of skill,  
I passe not for the rest,  
Good men except good will.

In—THOMAS GALE:  
*Certaine Workes of Chirurgrie,*  
London, 1586.

## PREFACE TO THE SECOND EDITION

There has been a steady progress in the field of cardiovascular roentgenology and this is witnessed by the many additions to the text. The captions to the new illustrative material are rather lengthy but this seems the only way to present cases in a properly integrated and correlated fashion. The new bibliographic references are indicated by asterisk.

The author owes very much to a number of physicians connected with Temple University Hospital and Medical School. Particular gratitude is expressed to Prof. W. Edward Chamberlain, head of the Department of Radiology, in whose department the roentgenologic studies were carried out; to Prof. Charles L. Brown, head of the Department of Medicine, in whose department the clinical and electrocardiographic studies were made while on cardiology service; and to Prof. Lawrence W. Smith, head of the Department of Pathology, and to his associates Drs. Edwin S. Gault, Ernest E. Aegerter, Augustin R. Peale, Machteld E. Sano, and Anthony L. Pietroluongo, all of whom made possible the anatomic studies.

For the privilege of using their illustrations, the author is indebted to Drs. K. Heckmann (Istanbul, Turkey), G. P. Robb and I. Steinberg (New York City), H. M. Stauffer and N. Learner (Philadelphia) and E. Zdansky (Vienna, Germany). Gracious permission has been given by Dr. M. J. Schlesinger and the C. V. Mosby Co., St. Louis, Mo. (Am. Heart J.) to cite in detail the method of injecting and dissecting the heart, and by Drs. G. P. Robb and I. Steinberg and Charles C. Thomas, Springfield, Ill. (Am. J. Roentg.) to cite in detail the method of opacification of the heart and great vessels.

Miss Fay McConkey, my secretary, has conscientiously, intelligently and patiently helped all along, and to acknowledge this is my sincere desire. Dr. R. V. Cohen has been kind enough to read and correct the captions, and Dr. I. W. Ginsburg has made helpful suggestions for the last chapter. The quality of the photographic illustrations is a high credit to Mr. W. J. Taylor.

H. R.

Philadelphia,  
May, 1942

*I look upon reviews as a kind of children's disease which more or less attacks new-born books. There are cases on record where the healthiest died of it, and the puniest have often lived through it . . . Attempts have frequently been made to prevent the disease by means of amulets of prefaces and dedications, or to color them up by personal pronunciamientos, but it does not always help*

GEORG CHRISTIAN LICHTENBERG

## PREFACE

The examination of the cardiovascular system by means of the roentgen rays is but one among several methods of study. The reader will realize that the application of one method sets very definite limitations to the desire to know and to understand the "whole." Thus only certain aspects have been covered and the space devoted to the different topics is not and could not be in proportion to their practical significance. Certain aberrations from the normal in the cardiovascular system can be demonstrated by roentgen studies only, a certain number are better diagnosed by roentgenologic than by clinical methods, while very many early changes can be elicited only by clinical methods. Often negative roentgen findings are as valuable as positive ones. To disregard this method means to renounce much of what our best trained sense, the eye, can perceive. The examination by roentgen rays has rightly been called an autopsy performed on the living. Its worth as well as its limitation must be evaluated by basic knowledge of the normal and pathological anatomy and physiology of the cardiovascular system on the one side, and of the fundamental, theoretical and applied physical principles of the roentgen rays on the other. Deficient training and deficient insight into problems will only lead to disappointment and to misinterpretation of results. It is inherent in any method that he who uses it tries to do as much as possible with it, and tends to overestimate its worth the more he knows of it or is limited to its use; and that he who cannot use a method tends to neglect and to underestimate it. In brief, this is the danger to the physician who is exclusively a roentgenologist and for the physician who has little or no experience with and no confidence in roentgenology. As late as 1920 the great master Sir James Mackenzie wrote: "Indeed I am doubtful if an X-ray examination of the heart has ever thrown the slightest light on any cardiac condition." (Oxford Medicine, II/2, p. 456).

Every roentgen ray study should start with a fluoroscopic examination. Rotating the patient during fluoroscopy has the same relation to a film record as a ride through the country has to a picture post-card of the same scenery. A film record in its frozen aspect expresses the statics, while fluoroscopy expresses the dynamics of the situation. The report itself should consist of two parts, one the description, the other the interpretation. One should center on structural and functional changes rather than attempt an etiological diagnosis. The beginner should strictly adhere to the rule of writing the report first, independent of the clinical knowledge of the case, and then correlate both findings. Knowledge of the clinical findings enables the expert to com-

plete the examination in a relatively short time, and special points of interest suggested by the clinical report can be studied more thoroughly.

Nature is a series of infinite variations and the application of a special method of study, roentgenology, furnishes convincing evidence that there is no single type of visceral anatomy and physiology. Normality, as a statistical concept, refers to the average or central values of the curve of variability; but many individuals who stray from these values are healthy, anatomically and functionally. Since there is such range of variation, errors in interpretation occur and organic disease is occasionally diagnosed when none is present, while one may fail to recognize such disease when it exists.

When the physician has in mind all the multifarious aspects, he speaks from experience in studying a new case. Experience of this type, however, cannot be transferred, hence the attempt to reduce to types and to present a statistical résumé. In this connection, it is perhaps not amiss to cite Armand Trousseau: "Of what use is this semblance of precision? When one of our colleagues showed the medical world the coincidence which exists between diseases of the heart and acute articular rheumatism, was that beautiful discovery received the less favorably because the discoverer said 'very often' in place of 'forty-four times in the hundred'? Was the influence of sulphate of quinine on miasmatic hypertrophy of the spleen less surely established when Bailly said 'almost always,' than if he had said 'ninety times in a hundred'?"

Of deficiencies in the presentation of the material, there must be many. To

satisfactory from the point of view of primary roentgenological approach. To give a description with purely roentgenological criteria as leading features, such as size, shape, position, pulsatory movements and contrast, becomes too analytic and calls for another synthesis which necessarily involves much repetition. The limitations of the middle way which has been adopted are obvious. Also, it seems to the author that never before has the attempt been made to integrate for one organ system, from a roentgenological point of view, anatomical, physiological, and pathological features, and experimental problems and clinical applications. It may well be objected that of these non-roentgenological facts either too much or not enough has been included. Yet, the time comes—and must come—when a work is brought to an end.

It may be remembered that it is not the method but the man who uses the method that decides its value. Arthur Schopenhauer expressed such a thought when he said "It is less important to see something new than to think something new of that which everyone sees."

Particular names, relating to any specific fact, have not been inserted in the text; not only would it have broken continuity but also it would have been impossible, in spite of repetition, to give credit to all contributors. Hence bibliography has been subdivided by appertaining chapters. The author has read in the original all articles cited in the bibliography, has abstracted nearly all of them, and will be glad to indicate to any worker in a limited field those articles which have a direct bearing upon his problem. Since a complete bibliography would exceed publication limits, many articles have, of necessity,

been omitted. All references are, directly or indirectly, of some significance for the roentgenologic approach, although this is not evident from some of the titles.

The beginner and all those without good visual memory will not derive all the available information from certain text passages, and this applies particularly to the first part of Chapter III, unless they refer to the illustrations, original roentgenograms and, in addition, a good anatomic model of the heart and the great vessels (see figures 9-11). The designation right and left on the roentgenograms, orthodiagrams and photographs refers to the patient; the designation right and left on the diagrams refers to the reader.

and Dr. Wilhelm Dressler in clinical cardiology. The rare privilege and fortune of being associated with Dr. W. Edward Chamberlain, Professor of Radiology, Temple University School of Medicine, can be realized only by those who know him personally. Dr. Chamberlain devotes much of his time and energy to the realization of his idea that the roentgenologist should hold the position of a consulting physician. It will give the author a great satisfaction if this treatise should be an aid in attaining this goal.

For the privilege of using their cases and illustrations, the author wishes to thank Drs. Paul A. Bishop (Philadelphia), John D. Camp (Mayo Clinic, Rochester), P. Cignolini (Genova, Italy), H. Dietlen (Homburg/Saar, Germany), W. Dressler (Vienna, Austria), R. Heim de Balsac (Paris, France), H. Holfelder (Frankfurt/Main, Germany), George W. Holmes (Boston), Chevalier L. Jackson (Philadelphia), Charles F. Nichols (Philadelphia), G. G. Palmieri (Bologna, Italy), M. Sgalitzer (Vienna, Austria), Merrill C. Sosman (Boston), E. Zdansky (Vienna, Austria). All other cases were studied personally both from the clinical and roentgenological point of view either in the Temple University Hospital, Philadelphia, Pennsylvania, or in the Herzhospital, Vienna, Austria.

The assistance of Drs. Mary H. Easby, Isadore W. Ginsburg, George C. Henny, Albert K. Merchant, Lester Morrison, A. E. Taft, Henry S. Thomas, William A. Thornhill, Barton R. Young, in the reading and correction of manuscript chapters, is gratefully acknowledged. Mr. W. J. Taylor has given his master technique to the photographic illustrations. Thanks are expressed for their most conscientious work to Miss Thelma Stevens in preparing the index, to Miss Sarah K. Ingram and Miss Catherine McElduff for secretarial work, and to Miss Marjorie Gerken for reading the proof. Thanks are also due to the publisher, Mr. Charles C. Thomas, for his interest and helpful cooperation. And the author hopes that others will feel, as he does, that the appearance of the book is a credit to its publisher.

H. R.

Philadelphia,  
May 1936

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## CHAPTER I

### TECHNIQUE

tube is centered on the screen the maximum opening of the shutter should not exceed the borders of the protecting leadglass screen. The tube and its frame must be well counterbalanced to permit an easy shift. The screen should always be kept in a plane exactly or almost exactly parallel to the plane in which the tube moves. The roentgen ray tube must be correctly centered, especially in relation to the opening of the shutter. A detachable crossbar may be placed in the course of the rays, its shadow should appear on the fluorescent screen exactly in the middle of the fluorescent area corresponding to the open space of the shutter, and the shadow of the bars should obviously be as small as possible. By using a Potter-Bucky diaphragm of the type constructed for fluoroscopic use, valuable improvement of contrast is obtained.

Orthodiagraphy consists in outlining the silhouette of shadows visible on the fluorescent screens by marking the orthogonal projection as obtained by the central ray, or, for practical purposes, as obtained by a small bundle of central rays. If one assumes that the distance of the tube anode is finite, which as a rule is 50-80 cm. for fluoroscopy, then, because of the divergent course of the roentgen rays, the image of any object will be larger than the object itself. If the central ray is moved tangentially along all silhouettes, the borders of all the objects examined (heart, great vessels, diaphragm, clavicles, etc.) will show silhouettes which are independent of the distance of the object as well as of the roentgen ray tube from the fluoroscopic screen. Special devices

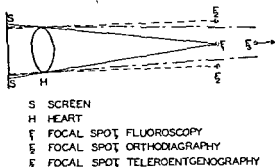


FIG. 1—Schematic drawing showing the relationship of heart, screen or film, focal spot, and the course of the roentgen rays, for the three methods routinely used in the roentgenologic study of the heart and great vessels

to be recorded by piercing holes in paper suspended from the wall at the side of the fluoroscope. The location of the central ray is represented by each puncture.





During fluoroscopy the physician is apt to be subjected to radiation that is mostly scattered since the primary beam is usually adequately limited by diaphragms and the screen is faced by lead glass. Exposure to direct radiation occurs chiefly, first, if the shutter is opened so wide that roentgen rays may pass outside of the fluoroscopic screen, and second, if the physician places his unprotected hands in the direct beam. If much fluoroscopy is done, one should consider the scattered radiation and wear an apron of lead rubber

The fluoroscopic image is dim. This arises from two circumstances. First, the fluorescent chemicals now available produce relatively little fluorescent light; only 4% of the roentgen ray energy that reaches the screen is converted into actinic light. Second, the amount of radiation must be limited to avoid excessive skin dose. Because of the dimness of the fluoroscopic screen the acuity of vision is much diminished, and adequate dark adaptation of the eye becomes necessary (this is discussed in chapter V). An increase in the ma, within certain limits, gives only a modest improvement in brightness. Increasing the voltage from 50 to 63 kv. doubles the brightness of the screen but the resulting moderate improvement in distinctness is likely to be of no value since the accompanying increase in scatter lowers the contrast. There is an advantage in increasing the object-screen distance. While the brightness diminishes, the contrast improves. This holds true in spite of the fact that the unsharpness increases.

**Roentgenography. APPARATUS** Three types of roentgen ray apparatus are in use. In one the roentgen ray tube acts as a rectifier as well as the generator of roentgen rays. The maximum delivery is 85 kv p, 100 ma. In another type one thermionic valve tube is used as a rectifier. The capacity is 100 kv p, 100 ma. Both types represent half-wave rectification, and the current flows through the tube during alternate half-cycles. These apparatuses are not recommended for chest roentgenography. The third type refers to the employment of four thermionic valve tubes for rectification. Here full-wave rectification is present and the tube current flows every half-cycle. This type has a capacity of 200 to 500 ma. and is recommended.

Apparatuses are heavy and costly because of the gross inefficiency of both the roentgenographic and fluoroscopic processes. The efficiency, i.e. the ratio of the useful roentgen ray energy to the electrical energy input from the high-tension generator to the roentgen ray tube, is only one ten-thousandth of one percent. The tubes must be designed to dissipate heat at a high rate.

In using the customary target-film distance of 180-200 cm, the size of the focus of the roentgen ray tube does not permit finest definition because of the necessary high load. In decreasing the size of the focus, one must reduce the target-film distance to 70-120 cm. At this distance the use of a cone is desirable. A tube with a rotating anode (Rotalix) gives very fine definition and allows for short loads, 7-8 times the energy permissible for a stationary anode. Such fine definition, as well as overexposure, is needed to demonstrate small lime salt deposits in the coronary arteries or in the heart valves. Fine definition is likewise desirable for a special study of the intrapulmonary vascular pattern.

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The simple procedure to be described here requires that the screen be independent of the tube. The screen is pressed tightly against the patient's chest while the tube is being moved from point to point. The shutter is narrowed so that there remains visible on the fluorescent screen an area of only a few square centimeters. The desired borders are then brought exactly into the center of this area by shifting the tube into the necessary position. The shadow of these borders is marked directly on the glass with a pencil. The largest possible silhouette is drawn, it corresponds to ventricular diastole for the heart area and to ventricular systole (discharge) for the large vessel area. The drawing is accomplished during quite shallow respiration. A hazily outlined left lower pole region is clearly visualized when the patient takes a deep breath. The contour should not be traced, however, until the patient has resumed quiet respiration. The contours of the diaphragm are sketched during the moment of respiratory pause, at the position of proximal (cranial) excursion. Care must be taken not to move the screen at any time while the outline is being drawn.

The use of an extra glass placed on the leadglass screen is to be avoided, for with increasing distance from the screen, an increasing error is introduced, caused by parallax. Further, drawing directly on semi-transparent paper placed over the leadglass is not advisable. Celluloid sheets (film base) may well be used, however. The drawing should be done by moving the pencil back and forth over the desired short stretch of the border in question. Then the tube is shifted along for a distance of 2-3 cm, the shutter opened moderately wide, the new portion of the border visualized, the shutter closed down again to a very small opening and the drawing continued. For example one may choose the following sequence: left clavicle, inner and upper left chest wall contour, left cardiovascular border, left leaf of diaphragm, inner and lower left chest wall. The procedure is similar on the right side. After completing the tracing, the tube is moved back along several short stretches of the outline at the left side. This check permits one to determine whether or not the patient-screen relationship has remained unchanged. It is instructive to draw the contours of the last posterior rib which is visible above the dome of the right leaf of the diaphragm as well as the outer skin contours. With metal marks, the epigastric notch and, in the male, the nipples can be visualized and drawn. The sketch is copied on semi-transparent paper for a permanent record. Drawings from oblique positions are done in precisely the same way. The position of the patient for the two typical oblique views is detailed in the chapter on roentgenography (see fig. 4). Orthodiagraphy may be used in either the vertical or recumbent position although the vertical is in more common use. The recumbent position is desirable for the examination of patients who are unable to stand or sit, or whenever a comparison is planned between the percussion findings, which have been obtained on the recumbent patient, and the orthodiagraphic findings. Orthodiagraphy is used on the same patient for both vertical and recumbent positions in order to determine varying degrees of filling of the heart. Such variations occur in quite a marked degree under certain conditions where, in the last analysis, gravity exerts an untoward effect on the distribution of blood in the body. A tilt table should therefore be an integral part of the fluoroscopic equipment.

up should also be chosen when a patient may not be very ill to begin with but where an aggravation may be anticipated because of the type of underlying disease. It will then be possible to have the standard film comparable with films taken at a later date. The diaphragm is easily displaced cranial in infants. Therefore the fasting stage should be chosen for them; and pressure on the abdomen, as exerted in the prone position, should be avoided.

It is desirable to know in which phase of the cardiac cycle the roentgenogram was recorded and this is indispensable when dealing with a comparison of heart or vessel size, when studying the relationship of the shape of the silhouette to the different periods of the heart action, or when the amplitude of the pulsations is to be measured. We not only need to know exactly and objectively at which moment of the cardiac cycle the exposure was made, but we must also be able to take an exposure at a certain predetermined moment (phase) of the cycle. Several methods by which this may be done are listed below.

1) by the use of the mechanical impulse of the heart (or the pulse wave in the carotid, brachial or radial arteries) The simplest method is to have the operator palpate the pulse and, when its peak is reached, close the exposure switch. The personal equation is obviously an undesirable feature of this procedure. A modification of this method consists of having the pulse thrust conveyed by air transmission to a tambour or a sensitive oscillogram against which a tiny mirror is suspended. The mirror reflects, in a certain position, an intense beam of light into the eye of a photoelectric cell. The current of this cell is amplified through vacuum tubes and is used to close the roentgen ray switch. In a more direct modification the tambour carries a platinum pin

ess of amplification; second, neither the apical nor the carotid thrust can be used in a number of cases; third, there is not an exact periodicity between the heart contraction and the pulse wave. In addition the distances from the heart to a stated point on a peripheral vessel varies in different individuals; and finally the velocity of the pulse wave is not constant, especially in such conditions as arteriosclerosis and aortic valvular disease

2) by the use of the heart sounds. The first heart sound begins at the onset of ventricular systole and during the moment of the presphygmic period the volume remains unchanged, representing the maximal diastolic size. The beginning of the second sound corresponds to the end of the period of ventricular discharge. During the moment of the postsphygmic period the minimal systolic size is obtained. While listening to the heart sounds, the key of the switch is closed (personal equation!), or a sound amplifying device can be used, automatically closing the primary current.

3) by the use of the differences in density of the heart shadow. Densitometry shows that the heart shadow undergoes regular changes in density with each cycle, increasing during diastole (filling with blood) and decreasing during systole (emptying of blood). The current of a photoelectric cell is amplified and registered by a string galvanometer or cathode ray oscillograph, and this graph may be further used (see 4.)

motion of the heart and of the tissues with the heart beat, and it increases in direct proportion to the exposure time within the limits usually used. It is therefore desirable to obtain a roentgenogram for the anterior view of an adult person, when taken at a target-film distance of 182-212 cm. (6 to 7 ft.), in one twentieth of a second or one thirtieth, if possible. Oblique and lateral views require two to three and one half times more exposure, other features remaining equal. This would require an undue increase in the load on the tube. It is therefore recommended to choose for these views a distance of 121 cm (4 ft.) and to increase the voltage, if necessary. The movement of the heart and lungs is least near the endpoint of diastole, and roentgenographic synchronization with this phase of the cardiac cycle produces a minimum of blurring. Second, the roentgen rays emanate not from a point but from a finite area, i.e., the focal spot of the tube. The resulting geometric unsharpness is proportional to the size of the focal spot. On the other hand, an increase in the size of the focal spot increases the load capacity of the tube and permits reduction of the exposure time. It is therefore necessary to compromise between unsharpness due to motion and that due to geometric unsharpness. The best results are obtained by means of a rotating anode tube. Third, films and intensifying screens produce unsharpness due to the characteristics of the materials of which they are made.

Two simultaneously exposed films are needed when an attempt is made to determine the heart volume roentgenographically. In front of and at the side of the subject are placed two films at right angles. Corresponding to these are two 40 k.w. rotating anode tubes placed at a target-film distance of 2 and 1.5 m. respectively. The two tubes are coupled in parallel, the high tension is 90 kv p., the tube for the front view has 200 ma. and the tube for the side view 250 ma. The exposure time is one-twentieth of a second, and the exposures are made simultaneously. In order to cut off scattered radiation and thus prevent blurring, two crossed all-metal grids are used in both of the planes.

If a high speed Potter-Bucky diaphragm is used and synchronized with the exposure, exposure factors are kept constant except the voltage, which is increased by 15 kv.

To estimate the volume of the heart for the erect and recumbent positions: Patient places hands over head. Mark skin points for subsequent central ray entry corresponding to ant. axillary line at rt. side and to midline in back, both at same level and about 8 cm. above level of nipples. All roentgenograms to be taken with patient holding hands above head and at end of tidal respiration, moderately overexposed, if possible at 6 ft. otherwise at 4 ft. focal-film distance. For erect position. Patient stands 8 min., then ant. and lt. lateral views. For recumbent position: a) Patient lies flat on his abdomen for 8 min., roentgen ray tube above, film below; b) patient lies flat on his back on a flat bench or table, horizontal course of roentgen rays from rt. to lt., film at patient's lt. side.

Chest roentgenograms may be taken with the patient in any position. The upright position is chosen by preference. For gravely ill patients roentgenograms are best obtained as posterior views, i.e., the film is under the patient's back, and the roentgen ray tube is fixed overhead to the ceiling. Such a set

over potential. When the control switch is closed, the plate circuit of the thyrotron is completed and a condenser, which by its charge had depressed the grid potential, begins to discharge. The grid potential thus rises and the thyrotron tube flashes over at the moment the grid attains the flashover potential. Immediate action of the roentgen ray circuit takes place for the diastolic position. For the systolic position a time delay of 0.20 sec is introduced. A specially constructed timer is needed (Hirsch and Schwarzschild).

2. The action current of the heart is led off to an oscillograph amplifier. The oscillograph is combined with an electric motor which drives a constantly varying resistance and thus the cathode ray crosses the oscillograph's fluorescent screen. A clear registration of the R and T deflections is obtained by adjusting the amplifier. The motor is furnished with a gear and by means of this the movement of the cathode ray over the screen can be regulated so that each turn corresponds to one pulsation. The roentgen apparatus time relay is connected with a contact on the axis of the motor. This contact has a corresponding registered point in the course of the cathode ray. A manually adjustable contact in the circuit to the roentgen tube prevents exposure before the oscillograph is correctly adjusted. When the desired heart phase is obtained the manual contact is closed. When, in the succeeding round, the cathode ray passes the point of contact the roentgen apparatus time relay is closed. At the same time the investigator sees from the high tension deflection in the oscillogram that the setting is still correct (Jonsell).

When the mechanical impulse of the radial artery is used, the following statements, hold for the average normal case. To obtain the systolic ventricular phase, the exposure is made half-way on the ascending limb of the radial tracing; to obtain diastole, two-thirds down on the descending limb.

It may be added here that all mechanical devices work with a certain lag which should be determined, while electric transmission for practical purposes is lag-free.

The cardiovascular silhouette is, furthermore, influenced by the phases of respiration. Exposures should be best taken at the end of quiet expiration. Poor contrast in the lateral views may necessitate a deep inspiratory phase; routinely, however, this phase should be avoided. The normal shape and size is altered under such conditions, especially if the patient involuntarily increases the intrathoracic pressure. If roentgenograms are taken with an exposure time sufficiently long to cover several cycles of normal respiration, the largest possible silhouette for this type of respiration is obtained. It is desirable for certain scientific purposes always to have the same phase of respiration as well as to have objective control of it. Several methods for this purpose follow:

(a) a glass tube system is connected with the mouth of the patient. A mercury column is moved by the respiration and, at one or the other end of respiration, closes a current for the roentgen ray switch.

(b) the different positions of a spirometer are registered by a galvanometer which is coupled with another galvanometer registering the action current of the heart.

(c) a Politzer bag is held in position in the axilla. By rubber tubing, it is connected with a vertically suspended lever of a tambour tipped with a lead

4) by the use of the action current of the heart. The relation of the mechanical contraction of the heart to certain phases of the electrocardiogram is well established. The peak of the R deflection precedes mechanical ventricular systole; hence an exposure taken at that time will register the maximum of mechanical ventricular diastole. The interval between the peak of the R deflection and the moment of the maximum of ventricular systole is relatively constant, namely about 0.20 sec., and an exposure with time delay will register the maximum of ventricular diastole. The relation of the end of the T deflection to diastole is not quite constant. With a few exceptions the R wave is rather high and steep and is the one deflection of the electrocardiogram which should be used for timing. Special precautions are necessary because the electrocardiographic record is easily interfered with by the low tension 60 cycle alternating current as well as by the high voltage, pulsating direct current. The string of the galvanometer when affected by such induced currents shows marked vibrations and may easily move out of the field. Thus it is necessary to provide electrostatic shielding. The string galvanometer should be set up in a metal covered room, as far away from the roentgen room as possible. The patient may be protected by a grounded metallic foil. Even better is a metal booth with an aluminum window permitting the exposure from a roentgen ray tube situated outside. Ordinary electrodes may be used, but needle electrodes placed intracutaneously are preferable. The leads from the galvanometer should run in shielded cables and when entering the room they should cross at right angles the aerial wires carrying the high voltage. The disturbance caused by the low tension alternating current can also be well overcome by inducing a current of equal frequency and opposite phase into the string circuit. One type of interference with the electrocardiographic record is really desirable: the onset and duration of the discharge of the transformer (duration of exposure) causes, by induction, vibrations of the string and thus registers the exact time of exposure.

In order to obtain exposures at any desired period of the cardiac cycle a regulating delay clock is used. The duration of systole and diastole for different heart rates is known and can be easily determined from the electrocardiogram. If it is desired to obtain an exposure at ventricular diastole, one will use a delay of about 0.48 second for a pulse rate of 60; while for a rate of 120, the delay is about 0.35 second. Obviously, the presence of a regular heart rate is required when applying this method. With this type of technique, roentgenograms taken at different times show not more than 0.1 cm. difference in size. At the very beginning of the ventricular systole, during the presphygmic period, which lasts 0.05 to 0.08 of a second, no change in heart size may be expected. Whether a change in shape sufficient to be observed roentgenographically takes place is not known.

The most satisfactory devices to obtain roentgenograms at predetermined moments of the cardiac cycle work as follows. 1. The patient's leads are connected to an amplifier which is connected with a special cathode-ray tube. This permits the operator to select the most suitable lead. The output of the amplifier is fed into a trigger circuit that consists of a thyrotron tube and a condenser. The magnified deflections of the E.C.G. are impressed upon the grid of the thyrotron in such a manner that the peaks never reach the flash-

tion, the air enclosed in the lungs will be exposed to the pressure exerted by the force of retraction of the lungs in addition to the weight and elasticity of the chest wall. The resulting inadequate filling of the heart will make it appear smaller than it is actually.

Practical examples are given here as to how requests may be ordered, the first part stating briefly the diagnosis and, if necessary, the problem involved. The second part refers to the technical procedure.

1. a) Old rheumatic heart with mitral valvular lesion; b) Ant. view, 6 ft., at end of quiet expiration, moderate overexposure, rt ant obl. view, about 50° rotation, 4 ft; at end of quiet expiration, moderate overexposure, thick barium paste in esophagus.

2 a) Calcific aortic stenosis; b) rt ant obl view, about 15° rotation, 2½ ft.; lt. ant. obl. view, about 60° rotation, 2½ ft.; both at end of deep inspiration, overexposure, coned down, center on level of calcific deposits. Note: mark by fluoroscopy level of calcific deposits by skin points

3. a) Aneurysm or excessive tortuosity of descending thoracic aorta. b) rt. ant. obl. view of mid-thoracic spine area, Potter-Bucky diaphragm technic; lt. ant. obl. view, about 40° rotation, 4 ft, at end of quiet expiration, same with thick barium paste in esophagus

4. a) Hypertension and left ventricular failure To determine appearance of lung fields prior to and following treatment b) ant. view, 6 ft, at end of deep inspiration Note: follow-up film to be done with exactly same technic.

5. a) Glomerulonephritis, preuremic picture b) post view, patient recumbent, 6 or 4 ft, at end of quiet expiration. Note follow-up film to be done with exactly same technic.

6 a) Bronchial asthma and emphysema To determine influence of respiration on heart size. b) ant. view, 6 ft and lt ant obl. view, about 60° rotation, 4 ft., all to be taken twice, at end of both deep inspiration and forced expiration.

7. a) Severe degree of postural circulatory deficiency. To determine heart size for vertical and horizontal positions. b) mark orthodiagraphically skin points on back of patient (midline) and on right side of patient (center of cardiac shadow). I) Patient to stand for 8 min, ant. view, 6 ft., end of quiet expiration; lt. lateral view, 4 ft, end of quiet expiration II) Patient lying on abdomen on floor for 8 min, tube above, ant. view, 6 ft, end of moderate inspiration. III) Patient lying on back on bench for 8 min, tube facing his rt. side, horizontal course of rays, lt. lat. view, 4 ft, end of moderate inspiration. Note: Patient should hold hands over his head for both the fluoroscopic and roentgenographic procedures.

**VASOGRAPHY.** A contrast substance injected into the vascular system for the purpose of visualization should fulfill the following requirements: it should contain the greatest possible number of atoms of a heavy element to give sufficient contrast even in dilution. It should be water soluble and have, preferably, a viscosity similar to blood. It should not decompose in the body and should not precipitate with the blood or other constituents of the body. It should not cause local vascular wall damage or spasm nor remote, local, general, immediate, or delayed toxic effects. Elimination should be complete and within a relatively short time. It should not be painful when in-



ring; its neutral position is indicated on the film holder by a lead strip. During respiration the lever swings to both sides, away from its neutral position, and the operator makes the exposure in either phase of respiration—recording this on a film.

(d) in like manner a pin is fixed to a tambour and, at a certain moment, dips into mercury, thus closing the switch.

(e) the respiration is recorded on a kymographic drum and the moment of exposure is marked with a puncture caused by a little spark gap.

The roentgen ray tube should be correctly centered for the midline of the patient. The height chosen is either at the intersection of the large vessels with the right heart border; or at the intersection of the pulmonary artery and left ventricle on the left. The level is marked on the skin of the individual's back during fluoroscopy by orthodiagraphic procedure. The angles for oblique positions are determined in each instance during the act of fluoroscopy. If this, for any reason, is not done and oblique views are still desired, then the choice for the right anterior oblique view is  $30-40^\circ$  if the aorta is mainly to be studied, and  $50-60^\circ$  if the left atrium is under consideration. For the left anterior oblique view  $40-50^\circ$  is advisable for the study of the aorta and  $50-60^\circ$  for the study of the ventricular mass. For the right anterior oblique view, the patient holds his right arm above the head; the dorsum of the left hand rests on the crest of the left ilium and as the arm is rotated inward the shoulder and elbow are brought forward as far as possible. For the left anterior oblique view the left arm is on the head, the right arm rotated and brought forward. For this purpose the bisacromial axis should be more rotated than the axis through the hips thus causing a spiral-like rotation of the trunk.

For each given instance the roentgen technician should be given a statement of the problem involved and detailed instruction how to proceed. Otherwise disappointment will result. It is assumed for the following discussion that instantaneous exposures are used and that the technical data (target-film distance, time of exposure, kv, ma, view, including degree of rotation) will either be photographed on the film or entered in the patient's record in order to assure adherence to the same technic for follow-up studies. It is highly desirable to have roentgenograms taken at a given instant of the cardiac cycle, but this is done at the present time in only a few places.

Although it is customary to take roentgenograms at the end of deep inspiration and there are certain special problems for which this procedure is desirable (chiefly to enhance contrast), roentgenograms of the heart and great vessels should generally be taken at the end-point of quiet expiration because they will then record a more physiological state and will avoid an abnormal narrowing of the cardiac silhouette which follows an involuntary Valsalva test. When the patient is asked to hold his breath in a position of deep inspiration, it frequently happens that he more or less involuntarily increases pressure in his chest with his glottis closed. This situation is referred to as the Valsalva test, which results in an increased pressure in the chest accompanied by a rise of pressure of varying degree in the abdominal cavity. Venous blood flow to the chest and abdomen is hampered. Even with respiratory standstill in a position of maximal inspiration and without action of the muscles of expira-

is likely to give deficient and misleading results because of the associated vasospasm.

**ARTERIOGRAPHY.** In case thorotrast, diodrast, or ethyl-tri-iodostearate are used the intra-arterial injection requires preceding local skin and tissue anesthesia with a few cm<sup>3</sup>. of 0.5% procaine hydrochloride. Most of the other halogen compounds require a central type of anesthesia, general for the upper and lower extremity, spinal for the lower extremity only. Because of loss of muscle tone and fall in blood pressure, certainly these types of anesthesia may lead to stagnation in some provinces of the arterial tree. The peripheral arteries are injected either by the transcutaneous method or after exposure by surgical incision. The first method may be applied to the brachial artery in the antecubital fossa and to the femoral artery in the trigonum or in the adductor canal. The latter method is recommended for the common carotid, subclavian, axillary, and popliteal artery. Highly skilled surgeons have injected all these vessels by the transcutaneous route. The musculature of the extremity should be exercised prior to the injection to bring about a functional hyperemia.

The position for the whole upper extremity is abduction and supination, and for the whole lower extremity marked outward rotation. If it is a question of the forearm region alone, pronation or a lateral position may be chosen. A dorso-ventral projection is used for the visualization of the vessels of hand and foot. At least two exposures should be made with a short time interval between them. The desirable length of this interval depends upon several factors. It should be shorter when there is relatively little obstruction to the blood flow, when using halogen compounds and when the injection of the contrast medium is made under considerable pressure. It should be longer when there is considerable obstruction to the blood flow due to multiple and extensive arterial narrowings, when using the slower moving thorotrast and when the injection of the contrast medium is made under but slight pressure. The amount of contrast medium required is about 10-15 cm<sup>3</sup> for the upper extremity and 20-30 cm<sup>3</sup> for the lower extremity. The special technique of taking roentgenograms is not standardized. Hence two examples of procedure will be given, one (a) for the upper extremity (Mayo clinic) and one (b) for the lower extremity (Dimtza and Jaeger). Both groups used the transcutaneous route of arterial puncture and thorotrast as contrast medium. (a) A sphygmomanometer cuff is placed around the arm near the shoulder. As soon as arterial blood from the brachial artery pulses into the barrel of the syringe the cuff is rapidly inflated above the systolic blood pressure and the radiopaque material is injected. The needle is withdrawn quickly and the first roentgenogram is made. The cuff is now deflated quickly to the level of the diastolic blood pressure for a period of 2 to 4 pulse beats, to permit the injected material to be carried farther distally. Then the cuff is reinflated quickly to its previous pressure and the second roentgenogram is made. The procedure is repeated for a third exposure. (b) A 20 by 90 cm. film is used. The radiopaque material is injected into the femoral artery under a 1.8-2 atmospheric pressure, a special apparatus being used. The roentgen-ray tube is centered at the proximal end of the film and is tilted 15° distad; the target-film distance is 1½ m. Attached to the tube is a shutter which covers one-half

jected, and when a paravascular injection occurs, no severe inflammation should result.

The four main groups of contrast substances are: 1) Halogen salts in oil—lipiodol, an iodized oil, and l'ombre (a very fine emulsion of lipiodol, with lecithine, albumin, glucose, glycerine). 2) Halogen salts in inorganic form—strontium bromide—(10-30% solution), sodium iodide—(up to 150% solution). 3) Halogen salts as organic compounds. The brands which are given in the following table have an iodine content of 42-52% and a 25-40% solution is used

<i>American Name</i>	<i>German Name</i>	<i>Chemical Formula</i>
Iopax	Uroselectan	5-iodo-pyridon-2-oxo-N-acetate of sodium
Neo-Iopax	Uroselectan B	Di-sodium salt of N-methyl-3, 5 di-iodo-4-pyridoxyl-2, 5-dicarboxylic acid
Skiodan	Abrodil	Sodium salt of mono-iodo-methane sulphonic acid
Neoskiodan Diodrast	Per-Abrodil	3, 5 di-iodo-4-pyridon-N-acetic acid diethanolamine.

4) Thorotrast—Thorium dioxide sol—(25% solution). Thorotrast is radioactive. Its radioactivity is rather weak; 100 cm<sup>3</sup> of thorotrast is equivalent to  $1.24 \times 10^{-6}$  gm. of radium; its half value is reached in 10<sup>10</sup> years. The alpha ray activity of 25 cm<sup>3</sup> of Thorotrast has been found to be equivalent to a maximum of 1.0 microgram and a minimum of 0.5 microgram of radium

None of these chemical substances fulfills all of the above mentioned criteria. At present substances corresponding to groups 3) and 4) are used exclusively. Thorotrast would be ideal but possible late effects due to its radioactivity have made its use open to question. A 25% thorium dioxide solution and 50% abrodil solution are about equal in density; but the former gives actually better contrast because it spreads less rapidly (higher viscosity).

It is the molecular weight and not the iodine content of the contrast medium that influences the degree of radiopacity. Thus neo-iopax with a 51% iodine content and a molecular weight of 493 gives better contrast than does diodrast that contains 59% iodine but has a molecular weight of 427.

Ethyl-tri-iodostearate is a sol with 50% basic substance and 20% iodine content. This substance is not available in this country at the present time. Selective deposition in the liver and spleen is noted as a side effect; there the contrast reaches its maximum after about 1 to 1½ hours and subsequently diminishes until disappearance within about 24 hours.

Visualization of the aorta requires the use of a highly concentrated mixture of abrodil (85%—2/3) and sodium iodide (150%—1/3).

Vasographic films always should be preceded by a plain film of the same region. Stereoroentgenography should be used whenever possible.

It is very important to realize that vasography that is associated with pain

zontally. If the sound stops at the level of the clavicle or deviates into the external jugular vein, retraction and slight elevation of the arm is of benefit. The right heart is reached at a distance of about 60-65 cm., but roentgenologic control should be used. The organic halogen (iodine) compounds have not been found to deliver enough contrast. 8-10 cm<sup>3</sup>. of a sterile 120% sodium iodide solution, injected quickly, gives excellent contrast. Following the injection, a small amount of a physiologic serum or sodium chloride solution is injected to remove the rest of the contrast substance from the lumen of the sound. The sound itself outlines the course of the *superior vena cava*.

The best results in the visualization of the chambers of the heart, the pulmonary circulation and the great blood vessels have been obtained by the method of Robb and Steinberg. 70% diodrast is used, containing 49.8% iodine. The dosage is from 25-45 cm<sup>3</sup>. First, the circulation time for the blood from arm to lung, and from arm to head is determined; the former by injecting intravenously 1/2 cm<sup>3</sup> of ether plus 20 cm<sup>3</sup>. of salt solution (the beginning of the injection and the detection of ether on the patient's breath is recorded); the latter by injecting intravenously 0.3-0.4 cm<sup>3</sup>. of a 2% solution of sodium cyanide in saline (the interval between the start of the injection and the onset of hyperpnea is measured). Instead of sodium cyanide one may use calcium gluconate (causing a sensation of heat), saccharin or decholin (causing a sweet or bitter taste). Second, the skin over a vein in the cubital fossa is anesthetized with 0.2-0.3 cm<sup>3</sup> of procaine solution, nicked with a knife blade, and a 12 gauge needle with attached stopcock is introduced into the vein. Needle and vein are flushed with salt solution every three to four minutes to prevent clotting. Third, 20 cm<sup>3</sup>. of blood is drawn into the syringe filled partly with diodrast. The arm is raised to an angle of approximately 45° above the horizontal. The patient exhales forcibly, inspires quickly and the content of the syringe is injected in two seconds or less. In the emphysematous patient the injection is delayed for 1/2-1 sec. after the beginning of inspiration. The inspiratory position is held until the opaque material has arrived in the pulmonary arterial tree and the roentgenogram has been made. The inspiratory position is assumed again shortly before the time of opacification of the heart and the aorta. Roentgenograms are taken in the desired positions. Fourth, after the last exposure the syringe is disconnected and needle and vein are flushed with saline solution.

The following technical points deserve attention. For visualization of the superior vena cava and the right atrium the time of exposure is 1 1/2 sec. after the beginning of the injection. For the right ventricle and the pulmonary artery tree the time of exposure is 1-2 sec. less than the arm to lung circulation time, usually 3 sec., but it may be longer because of slow venous inflow. The time of exposure for the left ventricle generally varies between 6-9 sec., deducting 1-2 sec. from the arm to head circulation time; it may exceed 20 sec. The roentgenograms require 8-12 kv. more than is usually used.

The optimal solution of the problem lies in rapid serial recording of the various cardiovascular structures during passage of the diodrast, either by regular roentgenograms or by photographs of the fluoroscopic screen image.

Immediate effects of the injection are a sensation of heat, the blood pressure falls but this rarely exceeds 30 mm Hg, the heart rate increases by an average

of the beam at its source in one position and which when shifted covers the other half. The shutter is placed so that the distal half of the extremity and of the corresponding portion of the film is protected while the proximal half is exposed to roentgen rays following the injection. During the subsequent second exposure the position of the shutter is changed so that the proximal half of the film is protected. The developed film shows the arterial system of the whole extremity, with the exposure proper for both the thicker proximal and thinner distal portion of the lower extremity.

**VENOGRAPHY** The peripheral veins are injected either by the transcutaneous method or after exposure by surgical incision. The transcutaneous injection into the antecubital vein will show contrast medium partly in the basilic and partly in the cephalic vein. Since the latter vessel varies in size and point of entrance into the axillary vein, injection into the basilic is given preference when the axillary and subclavian veins are to be studied. 20 cm<sup>3</sup>. of diodrast or 6 cm<sup>3</sup> of thorotrast are the amounts then required. The transcutaneous injection into a vein at the great toe or at the dorsum of the foot will demonstrate the superficial veins while the deep veins may or may not be visualized. The deep veins of the leg and most of the thigh are best studied by exposing under procaine anesthesia the small saphenous vein about one cm. behind the outer malleolus. A fine needle is introduced, first a few cm<sup>3</sup>. of saline are injected and followed by the continuous injection of the radiopaque substance. The leg is in full external rotation and moderate abduction. A satisfactory filling of the proximal venous trunks of the thigh cannot be expected until after 60 seconds, and a longer period of time may be required if obstruction causes resistance to the rate of injection. 20 cm<sup>3</sup> of diodrast are used. The best results are obtained with the patient standing, sitting; or when lying down, and the chief interest is in the veins of the calf region, the lower leg should hang down vertically or at least the knee should be bent with the foot at the level of the trunk. Incomplete information may be obtained when the lower leg is horizontal, apparently because of incomplete mixture of blood and contrast medium on the basis of a difference in specific gravity; an erroneous diagnosis of thrombosis or block may then be made. When the object of investigation is the common femoral vein, iliac vein, or inferior vena cava, the large saphenous vein is exposed under procaine anesthesia at about the level of the junction between the upper and middle thirds of the thigh. A large calibre needle is selected, a few cm<sup>3</sup> of saline are first injected and followed by injection of radiopaque material to be completed in 5 seconds. A posterior view is obtained. 50% diodrast is recommended for contrast roentgenography of the iliac vein and inferior vena cava.

The veins are also visualized some time after an arterial injection; as a matter of fact, a fairly complete visualization of the whole venous system of an extremity can be obtained by this method only.

**CARDIO-PULMONOGRAPHY.** The right heart and the pulmonary arterial system have been demonstrated in the following way. Under local anesthesia a

fossa in the proximal direction. The patient sits with the arm and

there to two other flasks. One contains warm saline, the other the injection mass. First, the saline is forced through the coronary arteries until the washings come clear. Then the mass is forced into the vessels at a pressure of 15 cm. Hg. It has been previously made up by soaking 300 grams of finest gelatine for two hours in 1200 cm<sup>3</sup>. of distilled water. To this is added 1000 grams of finely powdered barium sulphate, 500 cm<sup>3</sup>. of distilled water and 2 grams of thymol. This is heated over a water bath until the gelatine dissolves and is stirred until the whole mass becomes a homogeneous milky fluid which is then filtered through two layers of Victoria lawn.

The second method depicts only the coarser branches and the visualization of gross narrowing and occlusion. It is performed at the time of the autopsy. A 200 cm<sup>3</sup>. leveling bulb is filled with mercury, the upper level of which should be 150 mm. plus one-half the thickness of the heart above the bottom of the tray. The tubing is connected to one of the cannulas which are inserted into the coronary arteries. With the stopcock of both cannulas open, the mercury is permitted to run into the first cannula until droplets appear at the opening of the other cannula. The stopcock of the full cannula is then turned off and mercury pressure is maintained for about 30 seconds. The stopcock of the cannula through which mercury was injected is turned off, the tubing disconnected, the heart lowered by its base, an hemostat carried through the aortic orifice and the heart jarred to facilitate the escape of mercury from the ventricular cavities.

Stereoscopic roentgenograms of the specimen are then taken with a fine focus tube.

The roentgenographic interpretation of the injected coronary artery system is very difficult because of the overlapping of the various planes of the heart. Even the stereoscopic view leaves one in doubt about anastomotic channels and occlusion of branches. Furthermore, no final decision as to the patency or occlusion of any vessel can be made from the roentgenogram alone. For instance, an occluded zone of the vessel may be calcified and the shadow of this calcium may simulate an injection mass in the lumen. It is necessary to dissect the coronary arteries open. But a satisfactory dissection of the injected vessels is almost impossible after the specimen has gone through the process of fixation.

Schlesinger's method overcomes these difficulties. The coronary artery system is injected by a multicolored radiopaque injection mass; the injected and unfixed heart is cut open in a special way and unrolled so that all the coronary arteries lie in one plane, and finally the colored coronary artery tree is dissected out. Details of the method are as follows. 1. The tinted radiopaque injection mass: 60 gm. of lead acetate and 172 cm<sup>3</sup>. of distilled water, heated to dissolve, filtered, and allowed to cool (solution A). 24 gm. of disodium phosphate and 190 cm<sup>3</sup>. of distilled water, heated to dissolve, filtered, and allowed to cool (solution B). 1.5 gm. of agar-agar are placed in a 2000 cm<sup>3</sup>. bulb flask and there are added in sequence 100 cm<sup>3</sup>. of solution A, 1 cm<sup>3</sup>. of 0.06% phenol red, 70 cm<sup>3</sup>. of solution B, 10% NaOH to first permanent pink tinge (about 21 cm<sup>3</sup>.), and the total volume is brought to 200 cm<sup>3</sup>. with distilled water. The mixture is boiled over a free flame with constant stirring until the agar dissolves. 8 cm<sup>3</sup>. of methylene blue or basic fuchsin in saturated

of 30 beats/min., dizziness and nausea are observed; occasionally pallor, marked hypotension and vomiting. Delayed reactions consist of an occasional urticaria or mild local thrombophlebitis.

Certain precautions must be taken when planning for such a study. Several liver disorders, nephritis and hyperthyroidism represent contraindications, and caution must be used in patients with heart disease and circulatory failure. When there is a marked drop in blood pressure, or an allergic tendency epinephrin should be given. The stomach should be empty. The radiopaque solution must be clear and warmed to body temperature. The sensations to be experienced should be described to the patient in advance.

Another way of registering the cardiac chambers and great vessels filled with radiopaque material consists in making multiple exposures of the fluoroscopic image in rapid succession. A motor rotates a new section of film in a camera into position automatically after each exposure. With a lense of  $f$  2.0 and roentgen factors of 85 kv. 150 ma., target-screen distance of 67 cm. (27 inches), rotating anode tube, it has been possible to obtain a minimum of ten exposures during an 8 second period of observation.

**AORTOGRAPHY.** The abdominal aorta has been injected at two different points; at the level of the 12th thoracic or the 2nd lumbar vertebral body. The needle is 12-14 cm. long, has an external gauge of 0.12 cm. and contains an obturator to give it sufficient rigidity. The skin point of entrance is 4 fingers width to the left of the midline. The direction of the needle is practically vertical to the skin surface. For the upper injection the direction is mesiad, ventrad and slightly cephalad, parallel with the 12th rib. When the vertebral body is encountered the needle is sufficiently withdrawn to permit its forward progress again in a direction tangential to the vertebral body. For the lower injection the direction is mesiad and ventrad. The procedure as well as the actual exposure is accomplished with the patient in the prone position. Pressure is brought upon the proximal region of either femoral artery. General or spinal anesthesia is required. The amount of contrast substance is 20-25 cm<sup>3</sup>.; this is injected at a rate of 4 cm<sup>3</sup>. per second. The injected substance is rapidly diluted to a concentration of approximately 5-6%.

**LYMPHOGRAPHY.** The lymphatic vessels and nodes can be demonstrated by injecting 1-2 cm<sup>3</sup>. thorotrast subcutaneously, subfascially or intramuscularly into the extremity of living animals (guinea pigs, rabbits, dogs); they are permitted to run about and roentgenograms are taken in regular intervals approximately four times during the first hour and four times during the 24 hours; the next roentgenograms are taken at five day intervals. This method has, in a few instances, also been applied to man.

The study of the blood supply to the heart is much enhanced by a roentgenographic study of the injected specimen. As much blood and clot as possible are removed from the cardiac cavities by washing. Flanged needles or cannulas are inserted into the orifices of the coronary arteries and tied in place by passing a ligature after the circumference of the vessel has been cleared by blunt dissection. Three methods are cited.

The first one is particularly useful in studying the fine branches and anastomoses. The specimen is kept on ice and the injection is made 45 hours after death to allow rigor to pass off. Compressed air is led to a Wolff flask and from

ously washed with 100 cm<sup>3</sup>. of warm physiologic salt solution injected at 15 cm. Hg pressure until no more bubbles arise. The injection mass, kept in 50 cm<sup>3</sup> tubes, is melted in a bath of boiling water, is cooled and kept liquid by immersion in the same bath as the heart. Both coronary arteries are injected simultaneously with the warm lead-agar mass at 15 cm Hg pressure, the red mass into the right and the blue mass into the left coronary cannula. The pressure in the left coronary artery is then reduced to zero, and that in the right coronary artery is kept at 15 cm. Hg. The process is then reversed, i.e., low pressure is maintained in the right coronary artery and high pressure in the left. The heart is then cooled in a bath of iced physiologic salt solution.

3. The dissection of the heart to flatten out the coronary arteries: The first incision starts in the pulmonary artery, opening up the pulmonary valve and right ventricle on a line just to the right of the anterior interventricular sulcus, and extending completely to the apex. The second incision starts in the aorta, between the right and left anterior cusps of the aortic valve, behind each of which is one of the cannulated coronary artery orifices, extends down about 1 cm. into the base of the septum, and thus divides again the pulmonary valve and its ring. The third incision starts at the inferior termination of the second incision, continuing anteriorly along the base of the interventricular septum to the upper end of the anterior border of this septum, and then along the anterior border of this septum, completely to the apex; thus separating the interventricular septum anteriorly from the ventricles. The fourth incision starts again at the inferior termination of the second incision, continuing posteriorly along the base of the interventricular septum to the upper end of its posterior border, and then along the posterior border of this septum completely to the apex, thus connecting with the third incision and completely removing the interventricular septum. The fifth incision starts at the middle of the free border of the anterior cusp of the mitral valve, this cusp is bisected, and the incision continued through the mitral ring, and through the aortic ring to separate the left aortic cusp from the posterior aortic cusp. The left atrium having been entered from below, the incision is carried parallel to the left side of the interatrial septum to and through the pulmonary veins to unroll completely the left side of the heart. The sixth incision starts at the junction of the anterior and the medial cusps of the tricuspid valve, dividing the tricuspid ring and continuing across the aortic ring to separate the right anterior aortic cusp from the posterior aortic cusp. The right atrium having been entered from below, this incision is carried parallel to the right side of the interatrial septum, to and through the superior vena caval opening to completely unroll the heart.

4. Roentgenography of the flattened out coronary arteries 5. The opening of the coronary arteries: The injection mass is a gray mush that is easily removed. The intima of the vessels is tinted red if reached by the mass from the right coronary cannula, blue if reached by that from the left, and purple if reached by both.

**Volumetric Reconstruction.** The patient is placed on a revolving chair; this is rotated about a vertical axis which must pass approximately through the center of the heart. The procedure for the two stage method is as follows. The target is at the ordinary distance, i.e., 60 cm. By central (not orthodiagraphic) projection 12 or more drawings are made of the cardiovascular





FIG 2—49 yrs, M History Out of reasonably good health the patient developed the classical clinical picture of cardiac infarction, the ECG showed broadened P deflections,  $Q_{1,2}$ , absent intrinsic deflection in  $CF_4$ , S-T slightly elevated in I and  $CF_4$ , slightly depressed in III Congestive heart failure developed and the patient died 12 days following the onset of illness

*Postmortem* The heart weighed 600 gms and was enlarged The epicardial surface was partly normal, partly white and hyalinized and partly covered with fibrinous exudate. The left ventricular wall was soft, particularly the ventral and apical portions Schlesinger's method was used for injecting the coronary artery tree and dissecting the heart. Almost the entire heart was supplied with the blue injection material from the left coronary artery The right coronary artery showed two areas where the lumen was nearly occluded (arrows) A branch of this artery (3) was traced by dissection over into (2) a branch of the left circumflex branch (1) A good sized branch of the left circumflex was occluded over several cms (arrow), and the distal portion of this vessel (6) was traced by dissection over into (5) another main branch of the left circumflex (4) The left descending branch was occluded about 3 cms from its mouth by heavy sclerotic plaques (arrow) and there was present a fairly recent thrombus Another small branch nearby was also occluded. Microscopic Corresponding to the area of the occluded right coronary artery, chronic epicarditis and fibrosis; corresponding to the area of the occluded branch of the left circumflex, fibrous thickening of endocardium, patchy myosclerosis, area of

alcoholic solution are added After heating for another minute the solution is strained through gauze, and 35 cm<sup>3</sup>. are distributed into 50 cm<sup>3</sup>. tubes; they are stoppered and preserved at room temperature. If the mass is to be kept in stock for any length of time, it is better to add the dyes after it has been remelted for use just before injecting it into the arteries. 2. The injection of arteries: The right and left coronary arteries are cannulated. The heart is warmed to 44° C. in a bath of physiologic salt solution and is kept at this temperature throughout the procedure Both coronary arteries are simultane-

sect, the former is cut through in a linear fashion and above this cut a small box is mounted on the ruler containing an electric lamp, the switch of which is held by the examinee.

posterior surface of the

to which the horizontal movements of this rod are transmitted. When the rod coincides with the most lateral point of the cardiac contour, the midline of the ruler is in the same direction and vertically above the tangent from the focus of the tube to the most lateral point of the cardiac surface. The patient is revolved through  $360^\circ$ . The electric lamp is switched on and off at regular intervals, all the time letting the shadow of the vertical metal rod coincide with the most lateral points of the cardiac shadow. The developed bromide paper shows a mass of lines crossing each other under obtuse angles, delineating the projection of the heart.

**Cinematography.** Two main methods are available. 1) Direct method. Here the roentgen rays act directly on the film; image and object are grossly identical in size.

In order to obtain about 16 images per second, theoretically one could use either a continuously moving or intermittently transported film. The former method is entirely impractical, for in order to keep the blurring at less than 0.1 cm. on a film 12 cm. in length, an exposure of  $1/1900$  sec. would be required. With one technique for the second procedure a film strip 10 m. long is drawn through the frame by rollers for a distance of 28 cm. while a mechanism of eccentric and connecting rods holds the two intensifying screens apart and releases a pair of brake shoes designed to prevent movement of the film during the making of each exposure. When the film has moved 28 cm., the rollers no longer grip it firmly and the brakes are instantaneously applied to the edges of the film. The intensifying screens are then forced together, and the new area of film is in place and ready for exposure. With a similar technique for this method a roll of film 24 cm. wide, perforated along the edges is driven by sprockets and guided between two intensifying screens. During the part of the cycle when the film is at rest, it is clamped between the intensifying screens and a shaft with gears is so connected to the shutter that at this moment the exposure is made. The back intensifying screen is then released, the film moved downward 18 cm., and the above cycle repeated.

Exposure to the motion film must be interrupted by a short time.

of the primary current emission continues for a short time. With the latter method the shutter consists, for example, of two lead discs placed closely together between the roentgen ray tube and the patient and rotated in opposite directions on a common axis. A segment being cut from each disc, the

four and six valve rectification, respectively. The last current may be compared with a continuous emission of light of the same color, the former with light that passes through all colors of the spectrum and, within a second, is

replacing the screen by the cardboard sections; the body of the patient by a block of plastic material, which rests on a revolving table; the tangential ray by a metallic wire, one end of which is fixed to a handle, the opposite end being attached to a point which corresponds in distance and height to the target. The wire is moved along a cardboard section and in so doing cuts off parts of the block of plastic material. With each cardboard, this block is rotated into

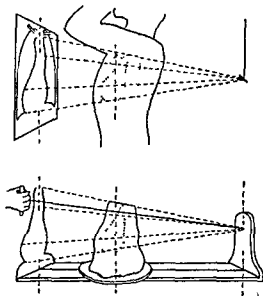


FIG 3—Roentgenologic volumetric reconstruction, method of Palmieri. By central (not orthodiagraphic) projection 12 or more drawings are made of the cardiovascular silhouette by revolving the patient through 360°. The different tracings are cut out in cardboard. In a second stage a wire is moved along the cardboard section and parts from a block of plastic material are cut off. With each cardboard, this block is rotated into the corresponding angle. (Adapted from Palmieri, G. G., *Arch Mal Cœur*, 1921, 14, 440.)

the corresponding angle. When the process is finished for each cardboard sil-

The indicator is fixed to two arms, movable at right angles to the rays and its elongation is formed into a metallic string, supported by a bow-like arrangement, and parallel in direction to the central ray. This wire cuts sections out of the plastic material corresponding with the delineated outlines on the screen. The model is complete when the patient has made a complete rotation. This method as compared with the former has the following disadvantages: the patient is exposed longer; a false movement with the indicator on the screen may destroy the model.

**Horizontal Orthoprojection.** In the prolongation of the axis of a revolving chair there is mounted above the stand, a horizontal wooden disk which ro-

turned on, the focal spot of the tube is at the level of the slit. The record is represented by a continuous band whose edges bear waves corresponding to the movement of the portion of the cardiovascular system exposed in the aperture. In the case of the heart border, the trough of the curve represents systole and the crest, diastole. In order to record the portion of the cardiovascular shadow of which the curve is made, a control film is completed before the kymographic record is started. One can use two independent lead screens and place one in front of the left heart contour, the other in front of the right heart contour. Or, again, one with a transverse (horizontal) opening can be placed over the heart, the corresponding film moving vertically. Another lead shield with a transverse (vertical) slit is placed over the level of the right leaf of the diaphragm, the corresponding film moving horizontally: thus pulsations may be recorded which are differently directed. When the slit image is photographed on the moving film by the rectified full wave 60 cycle current, impulses are registered as alternate light and dark bands and can be used as timing marks.

**Multiple Slit.** To comprehend the movement of the organ as a whole it is sufficient to register the individual movements of parts which are separated by regular, relatively short distances; the movement as a whole may be deduced from the coordination of the single movements. The exposure given each point of the film is indicated by the ratio of the slit width in millimeters to the film speed in millimeters per second. The intensity of exposure must be uniformly maintained throughout the entire time of exposure and constant potential should be used. The above mentioned ratio (i.e., the equivalent single exposure) should be small,  $\frac{1}{16}$ – $\frac{1}{30}$ .

a) The lead screen has many equally spaced, horizontal slits. The length of the record of the movement of the particular part, exposed in the various apertures, is necessarily limited in order to avoid overlapping of the records. Exposure and movement of the film-holder is, therefore, limited to the time necessary for the film to move slightly less than the distance between the openings. The technical details of the method vary with different authorities: size of the slit 0.04–0.2 cm., rate of film movement 1–2 cm. per second, duration of exposure 1–3 seconds; target film distance 70–150 cm. In addition to the kymogram a control exposure is made to afford a basis for the measurement.

cranially or caudally. In the former case, the record is to be read from above down; in the latter, from below up. Further, one may turn the film 90° and then read the record along a horizontal line, in the customary manner. The kymogram differs from the ordinary roentgenogram. First, it is crossed by regularly spaced lines dividing the film into a series of horizontal strips, as there were slits in the lead screen.

as the film moves

in each bar, and crossing it, several waves are noted, they correspond to the line of demarcation between the shadow cast by the cardiac silhouette and the transparency of the lungs.

upon the cardiac rate and the

bar the projection of the movement of a very short portion of the silhouette is

extinguished many times. A six-valve rectified current (three phase constant potential), at 0.01 second exposure, equals in precision a four valve rectified current at 0.25 second exposure and a one valve rectified current at 1.0 second exposure. The current, obtained by four valve rectification, approaches the constant potential graph when long, metal shielded and grounded cables are used; this condenser effect diminishes rapidly, however, for currents stronger than 50 ma.

2) Indirect method Here the screen image is directly photographed with a cinematograph camera, the image is therefore much smaller than the object.

In order to record an image on a cinematographic film in the fraction of a second certain technical requirements are necessary. The screen preferably should emit a bluish light, and the film should be sensitive to this particular wave length. The camera lens must have as large an aperture as possible to allow the maximum amount of light to pass through it. In a recent study an f.0.85 (Zeiss R-Biotar) lens was used. The distance from the screen to the front of the camera was 158 cm. and a field of  $25 \times 45$  cm. was covered. Difficulty was experienced in obtaining an accurate focus of the screen image; in order to overcome this, a metallic grid was placed just behind the screen and used as an object to be photographed when illuminated by the roentgen rays. Other factors in this study were: a 20 kw Metalix tube (a 10 kw Metalix tube with a focal spot of  $4.1 \text{ mm}^2$ . was found sufficient for all requirements); 0.5 mm. aluminum filter; 70 ma, 120 kv max.; Ilford SX screen, Gevaert orthochromatic film. 7 exposures per second were obtained, each lasting  $\frac{1}{14}$  of a second. In order to protect the patient from overexposure a synchronizing switch was used in the circuit; a rotary contact breaker was connected with the driving spindle of the camera mechanism so that the roentgen ray tube was active only during the period of time that the camera shutter was open; since the time of motion of the film and the time of the stationary period are of approximately equal length, the dosage is by this means halved.

Further progress has recently been made by using a group of lenses, one of which has an aspheric surface resulting in an f.0.53.

Most instructive data are obtained by recording the passage of opacified blood through the heart and great vessels. 30 to 40 cm<sup>3</sup>. of 70% diodrast, injected intravenously, is used as a contrast medium.

In taking a cinematographic film, it is necessary to obtain only one complete cardiac cycle. The resulting negative can be printed off again and again on a positive film and a cinematic (movie) reel is thus obtained without exposing the patient too excessively to the roentgen rays.

**Kymography. Single Slit.** Movements of organs which are visible on the fluoroscopic screen may be graphically registered. Kymography is a spatial representation of movements which occur in chronologic sequence. A typical technical arrangement is as follows. In front of the patient is placed a lead screen which can be adjusted in height. This screen has a transverse (horizontal) slit 0.3 to 1.0 cm. in width. The level of the aperture is adjusted over the pulsating heart or vascular silhouette as viewed by fluoroscopic study. On the other side of the lead screen is a film holder which, by means of a motor, is made to travel at an equal given speed (4-5 cm. per second) at right angles to the direction of the opening. While the roentgen ray is

rate of film movement is 5 cm. per second; the duration of exposure 12 seconds; 0.1 cm. film corresponds to 0.02 second. Since the film moves at high speed and since, in addition, more cardiac cycles are included, the graphic results are very satisfactory.

**Kymogram and Heart Sounds.** The heart sounds are transmitted from the chest wall into a microphone, the output of which is amplified by vacuum tubes. An oscillograph deflects a lead marker which is roentgenographed on a small film and which moves synchronously with the kymographic film.

**Densitometry.** The heart varies in thickness during its cycle and therefore absorbs varying amounts of roentgen rays. By placing an ionization chamber in front of the precordium the resulting variation in intensity of the transmitted rays may be registered. From this record the varying thickness of the heart can be determined. One may mark on a finished film points of equal density. By connecting these points with curves, lines of equal density are obtained, they are called isograms. In addition, the densest area or the center of gravity may be determined.

As compared with mechanical devices, all roentgenologic registration works practically free of lag.

An evaluation of these different methods, their results, and limitations, is found in chapter V

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graphically demonstrated; in two adjacent bars, movements of two contiguous areas are represented. Wavy lines, caused by the movement of the pulmonic shadows, are seen farther lateral. Third, the shadows are not of homogeneous density, the varying density of the heart during its cycle being recorded.

The cardiac movement can be reproduced by observing the kymogram through a grid moving at approximately the same rapidity as during exposure, with a rotating glass-paralleliped in front of the eye.

Time periods are recorded during the exposure and permit the analysis of

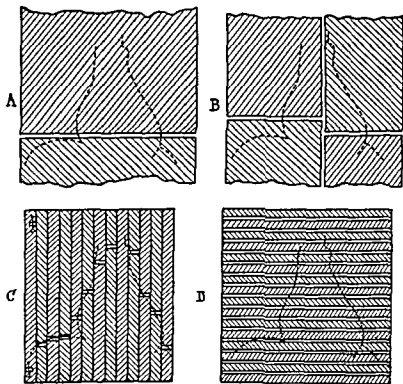


FIG. 4—Kymographic methods A—single slit B—single slit, modified C—multiple, interrupted slits D—multiple slits. From *Rontgenchirurgia cardiaca e regmografia*, Bologna, 1934, p. 2. Courtesy Prof. Dr. P. Cignolini, Genova and L. Capelli, publisher.

time relationships. By marking a definite distance corresponding to a certain fraction of a second on the waves of each bar and connecting these points, the outline of the entire heart at any particular instant may be drawn.

b) The grid diaphragm consists of a frame with 12 lead bars, each 24 cm. in height and 2.3 cm. in width. Each one contains a horizontal slit 0.2 cm. wide. If the removable bars are arranged in the proper order, the slits are evenly spaced 1 cm. apart. Under fluoroscopic control these bars are so placed that the slits overlap those points of the cardiovascular silhouette which are chosen for graphic registration. The film represents 12 vertical bars; in the more centrally located bars, to the left and to the right of the midline, appear the kymographic records of the different chosen points of the silhouette. The

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## CHAPTER II

### ANATOMY AND ROENTGENOLOGY

**Anatomical Facts.** A review of certain anatomical facts promotes a better understanding of some of the roentgenologic findings.

**HEART.** The heart weight in the newborn is on an average 0.76% of the body weight. Toward the end of the first year an average ratio of approximately 0.46% is reached and maintained from then on. This relationship between heart weight and body weight holds true as long as the body weight expresses a normal, proportional development. By the end of the first year, the left ventricle has grown proportionately more rapidly, the circumference of the aorta has almost tripled and that of the pulmonary artery about doubled. The chest gradually becomes flatter from birth through childhood. The form of the heart changes with age. In the newborn, the heart is short and plump, and the atria are proportionately large, especially the auricular appendages. The right and left ventricles are almost equal in size. The thymus pushes the base of the heart dorsad producing a rather horizontal course of the large vessels including the ductus arteriosus. In the second half of the first year, a definite descent of the thoracic and abdominal viscera changes the position of the heart. In children, the inflow tract predominates over the outflow tract; in other words, the distance from the auriculoventricular orifice to the apical pole is greater than the distance from the latter to the aortic and pulmonic orifices.

In the aged, the ring of the heart base is wide, the inflow tracts are elongated. The lower position of the diaphragm stretches the mediastinum vertically.

The ratio of 0.46% previously mentioned shows an average variation of 8% from the mean figures. More extreme variations are encountered in the presence of unusually high or low body weight. There is no proof, as anatomy

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of 45-55 years; this, how-  
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by the inclusion of hypertensive cases. At 60 or 65 years of age, atrophy begins and the actual loss of weight of the heart may be as much as 50 grams.

Normal hearts show definite variations in their general shape, independent of their volume. It can be seen by simple inspection and can be demonstrated by postmortem measurements that the normal average heart fades out into two types: one an elongated heart and the other a short, globular heart. The average of the entire posterior diameter of the heart  
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twice as much fat is found over the right heart as over the left, the weight of the fat is 5-30% of the heart weight and even higher in the older age groups. The hearts of many obese people show evidence of hypertrophy and, in the majority of instances, the weight of the heart plus the gross epicardial fat is absolutely greatly increased while the ratio of this weight to the body weight is often decreased.

The size of the heart should be expressed by both weight and volume, the latter referring to the sum of musculature and cavities. The weight of the musculature, expressed in grams, exceeds its volume, expressed in  $\text{cm}^3$ , on an average by 7.4%. Anatomic statements as to the volume of the heart usually refer to the capacity of the heart chambers only; in addition, one must remember that postmortem changes alter the size of these cavities. Such capacity determinations give the following results: about 30% of the total volume for the right atrium, 24% each for the right ventricle and left atrium and 22% for the left ventricle. In a series given by two investigators, the proportion of muscle weight of a chamber to its volume was found to be about 10-13% for the right atrium, 55-68% for the right ventricle, 9-18% for the left atrium and 104-110% for the left ventricle, there are, however, considerable variations from the average figures. With changes in the heart volume the surface, of course, changes in a lesser proportion, since doubling the volume of a sphere requires less than twice its original mass of musculature while a sphere of half the original volume would require more than half of the original mass of musculature.

The position of the heart with respect to the antero-posterior dimension of the thorax is somewhat variable. In the average case and at the level of the 6th thoracic vertebra, it may be assumed that the main axis of the heart corresponds approximately to the juncture of the ventral and central thirds of the chest.

The esophagus lies close to the dorsal surface of the pericardium (heart).

**AORTA.** Normal variations and changes brought about by age constitute our chief interest.

The normal aortic arch changes its direction. In its ventral-proximal portion, from the origin of the innominate artery to the left border of the trachea, its course is moderately oblique. In its dorsal-distal portion, it is almost in a sagittal plane. More ventrad the arch is in direct contact with the left border of the trachea. Farther dorsad, where it begins to descend, a close relationship to the esophagus exists. The esophagus has at that level an indentation, called the aortic arch impression.

In children and adolescents the aortic arch is situated in a slightly oblique plane, i.e., it does not present the kinked arrangement of the adult aorta that permits of a differentiation between a frontal-pretracheal and sagittal-paratracheal course. In the adult the trachea comes to lie in front of the esophagus, but the trachea in the child is farther to the right, and thus the tracheo-aortic distance is greater than the esophago-aortic distance. Very gradually the trachea moves from right to left and it is apparently due to this change in position of the trachea that the aorta shows the kinked course in adult life.

The dorsal-distal portion of the arch holds an oblique course in the aged. The descending thoracic aorta has a prevertebral position in its caudal portion in the adult. In the aged its position is almost entirely paravertebral, sometimes with an accentuated convexity dorsad and to the left.

The degree of elasticity is inversely proportional to the age. Or to express it differently, the product of elasticity and age is constant. Elasticity is used here in the sense of the power to yield and to resume original form. High elasticity



means that marked pressure changes cause marked volumetric changes. The change in the retractive capacity of the aorta has been estimated to be as 4:2:1 for the ages 25:45:65.

The aortic size increases continuously with age, and while the average circumference of the aortic root for the age of 20-30 years is about 5.9 and 5.5 cm. for male and female respectively, the figures for the ages of 70-80 years are 8.2 and 7.7 cm. To give another figure as expressed by one writer, the average circumference increases about 20% from the age of 18 to the age of 50 years. Considerable variation is, however, encountered. The figures given by different authorities for the circumference of aortas studied in a large number of individuals who were either killed during the war while in good health or who died by suicide or following a disease of short duration all point to the same result. Variations of 20% and 30% from the mean figures in 10 and 30 year age groups respectively, differences of 2.5 cm. and such extremes as 100% in 10-year age groups, extreme figures of 4.6 cm. and 7.1 cm. in the group between 18 and 38 years. In people with high body weight, the aorta is found to be above the average size, while body height is no correlation factor.

The concept of the small aorta (aorta angusta) as an anatomico-clinical entity should be discarded.

It was found for a large number of animals that the aortic cross section varies approximately as body weight<sup>0.72</sup> and as heart weight<sup>0.8</sup>.

**PULMONARY ARTERY AND VEINS.** The pulmonary artery increases in size with age as does the aorta. The average circumference increases about 11% from the age of 18 to the age of 50 years. On account of the slower increase in size, it is surpassed by the aorta near the age of 45. The pulmonary artery points to the left, and its T shaped bifurcation meets the inverted Y bifurcation of the trachea. The pulmonary artery divides at the level of the cranial edge of the left bronchus about 2 cm. below the tracheal bifurcation. The main right pulmonary branch bridges the angle of the tracheal bifurcation, crosses the main right bronchus behind the sup. vena cava, then leans its caudal-dorsal border against the cranial-ventral border of the bronchus, and both structures show a spiral twisting which puts the artery on the outside of the bronchus, the situation which it occupies in the hilum, and artery and bronchus make an arc equal to an eighth of a circle that overhangs the left atrial cap. The main left pulmonary branch arches with a left caudal concavity, which rises in front of the main left bronchus at about 1 cm. above the level of the right pulmonary branch and lies in its subsequent course more dorsad, caudad and to the left. The left (as well as the right) bronchus holds a slightly dorsal course, and the esophagus at this level tends to deviate slightly ventrad. Hence the root of the left bronchus frequently tends to cause a shallow indentation on the esophagus that is oblique from cranial-ventral to caudal-left-dorsal and 2.5 cm. in length. A uniform distribution scheme for the course of the main bronchial branches cannot be given, they show considerable variation. Bronchial canals closely accompany the arterial subdivisions within the lung parenchyma. These subdivisions may reveal two extreme types. The one shows long main branches giving off smaller branches at rather sharp angles, the other is characterized by short main branches, the angulation being less sharp. As a whole the degree of angulations decreases from the hilus toward the periphery. The distribution for

each lobe is rather characteristic; on the right side are found 3 main branches for the anterior (upper), 1-2 for the middle and 2 for the posterior (lower lobe); on the left side 5 for the anterior (upper) and 2 for the posterior (lower) lobe. The veins have another type of distribution. The main trunks are short and in the immediate vicinity of the mediastinum. Even at the lung root, they represent a complete set of diverging and quickly ramifying branches resembling the fingers of an outstretched hand. Some of the larger veins correspond in their course to the arteries and cross each other at the hilus; but generally bronchi and arteries on the one hand and veins at the other have an alternating course, they cross each other obliquely near the hilus, even perpendicularly in the periphery. Nowhere do the larger pulmonary veins approximate the size and thickness of the larger pulmonary arteries.

In the newborn the lesser circulation, as represented by arteries and veins, is comparatively feebly developed on account of the fetal inactivity of the lesser circulation. However the trunk of the pulmonary artery is well developed. As compared with the adult, the pulmonary artery and aorta are further apart, i.e., separated by lymphnodes and mediastinal tissues, but they are noted to be more in juxtaposition.

**SUPERIOR AND INFERIOR VENA CAVA.** A marked variation exists as to size. For the age groups 30-80 years, 40% for men and 20% for women was found.

**PERICARDIUM.** A triangular veil-like fold of mediastinal pleura extends from the lateral left caudal surface of the pericardium to the diaphragmatic pleura. Usually it approximates the left interlobar fissure. After puberty, fat is found along its ventral surface. No rigid attachment exists between the free anterior pleural portion of the pericardium and the posterior surface of the sternum.

The pericardium usually covers about 5 cm of the pulmonary artery and about 6-8 cm of the aorta, up to a point near the origin of the innominate artery. The ligamentum arteriosum, which extends from the aorta to the pulmonary artery, is covered in part or entirely by pericardium, in the latter case it produces a fold, clearly visible from the outer aspect. The line of reflection of the pericardium shows considerable variation at its cephalic aspect. The dorsal aspect of the inferior vena cava is extrapericardial.

In the subserous layers of the pleurae, especially in front of the lower portion of the pericardium, fatty-serous pads, fringes and folds are found. With the exception of the first few years of life, they are present in all age groups. They persist in cachectic stages, their fat content is less marked in young and their

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though not regularly, there is a pad of fat at the right side and also extrapleural, in front of the ascending aorta.

**Comparison of Anatomical and Roentgenological Findings.** The size and shape of the heart and of the large vessels change after death. These changes are partly exogenous, partly endogenous in nature. Exogenous changes are: a) a diminution of the elastic traction of the lungs on the pericardial surface at the moment when the chest is opened, b) elevation of the diaphragm. Endogenous changes are caused by the change in the myocardial tonus and by

the disappearance of the blood pressure. The myocardial tonus has already diminished during agony. The heart stops, with a few exceptions, in diastole; an immediate dissection shows the heart to be definitely larger than it is found to be later. The postmortem contraction may be complete or incomplete. With instantaneous death and with healthy heart muscle its onset is marked and persists for a long time. The different heart chambers contract to different degrees, depending upon the development of the respective muscle layers. For the normal heart, it can be stated that during rigor mortis the left ventricle empties nearly all its blood into the empty arteries, and perhaps some into the left atrium, while the thin-walled right ventricle does not expel an appreciable amount of blood into the lesser circulation which becomes engorged with blood during agony. Hence, the right ventricle is found to be rather wide while the left ventricle is reduced in size. The heart therefore decreases in its transverse diameter and appears more pointed. When rigor mortis subsides the heart muscle relaxes, the blood which meanwhile has coagulated does not flow back from either the aorta or the left atrium. Not later than 24 hours post-mortem a secondary and final dilatation takes place. Investigations have shown that rigor mortis may be less marked or even absent with higher degrees of myocardial degeneration, when individuals die of prolonged febrile disease, or with high fever of short duration and with marked anemia.

Experimental filling of the heart with contrast substance distends the heart and vessels because there is no tonus to resist. The mass of clotted blood in the cardiac cavities interferes with filling and varies from one case to another. To cite two extremes: *nothing is found when death occurs from a hemorrhage*, but much is noted when it is due to pneumonia. Also, the proportion of the width of the large vessels to the width of the heart as compared with the living is liable to change in favor of the vessels with these filling experiments.

The absence of the distending force of the blood pressure and of the tonicity of the smooth musculature is responsible for a decrease in diameter and length of the aorta, and is more so in the young than in the aged. If the aorta in dogs is inflated to a pressure of 175-180 mm Hg, it can be seen to increase in length as well as in diameter. And it has been shown for dogs that the aorta decreases 5-12% of its diameter during the first 12 hours postmortem. Filling experiments on cadavers show the border of the ascending aorta inside the right border of the sternum and separated from the shadow of the superior vena cava by a free space. If, however, the pressure in the aorta is increased, for instance by inflating air, then the right border of the aorta approaches the right caval border, without, however, quite reaching or surpassing it.

Fresh postmortem specimens have been filled with varying amounts of water; the resulting different volumes were determined and the corresponding roentgenologic projections obtained by orthodiagraphy. In order to obtain the volumetric changes, expressed in  $\text{cm}^3$ , it was necessary to multiply the changes in area expressed in  $\text{cm}^2$  by a coefficient which varied from 9-14.

The poor air content of the lungs after death interferes with the visualization of the outlines of the cardiovascular system, and artificial inflation easily leads to leakage.

The discrepancy between the findings in vivo and postmortem is further marked when we consider certain pulsatory phenomena. Injection experiments

show the left atrium and auricular appendage to be entirely at the dorsal aspect of the heart; whereas the left auricular appendage may be seen in the living when the left ventricle empties and the heart rotates. The injection of a contrast substance in the living dog shows systolic distention of the infundibulum which is found to be absent in the animal sacrificed immediately after the injection experiment.

It is not possible to distend the pericardium appreciably in the cadaver. In the living, however, a considerable stretching does occur, provided ample time is available.

Our knowledge of the silhouette of the cardiovascular system as well as of its inner topography is derived, in part, by comparing the results of the roentgenologic examinations of cadavers, preferably in several positions, with those obtained by actual anatomic dissection. The following methods have been used:

- 1) a roentgenogram is made and the silhouette borders are compared with the subsequent postmortem findings
- 2) long pins are thrust through the chest wall, a film is made and after opening the thorax the points are noted where the pins pierced the heart and large vessels
- 3) following formalin fixation, parts of the anterior chest wall are removed, the pericardial cavity is opened, and differently denticulated and punched strips, rings, and other pieces of lead are sewn to different portions of the heart and great vessels, thus designating the coronary and interventricular sulcus, the apical portion, the atrioventricular rings and semilunar valves, aorta and pulmonary artery. The pericardium is sewn together, the removed portions of the chest wall are replaced and roentgenograms are made in different positions.
- 4) after formalin fixation and freezing, the chest is cut into frontal sections, the cuts being parallel with the anterior surface of the chest. At each section points or areas of interest are marked by pieces of metal or by lead paint. Each single section as well as the superimposed sections can be roentgenographed.
- 5) the cavities are rinsed to clean them from bloodclots. All the orifices are closed with cork stoppers of appropriate size. Those in the topmost orifices (superior vena cava and one pulmonary vein) have two holes with a short glass tube in each; those in the lowest orifices (inferior vena cava and one pulmonary vein) have one hole with a short glass tube in it. The heart is vertically suspended in cold water and liquid gelatine (temperature  $40^{\circ}$ - $42^{\circ}$ ) is poured into one hole of each of the top influx tubes while the air escapes through the other top outflux tubes. When this is accomplished, the pressure in a manometer that is connected with the afflux tubes is raised to 8 cm. water. Finally all tubes are closed and the gelatine is allowed to solidify. Roentgenographic studies and actual measurements are then carried out on the specimen.
- 6) (See fig 18-21); the right and left cardiac cavities including the central veins and arteries are injected with a contrast medium. The following technic is suggested. After a median incision on the neck of the cadaver and ligation of superficial veins, the right internal jugular vein and common carotid artery are cut and their cephalic ends ligated. A metallic, slightly curved cannula with threaded surface is introduced into the proximal portion of the vein and a tight ligature is placed around it. A soft urethral catheter of adequate size is introduced into the artery, and is pushed forward until its tip has passed the aortic valves. It is then fixed by means of a tight ligature. A rigid sound or trocar may have

to be used in order to penetrate resistant aortic valves. The left cervical neurovascular trunk is now doubly ligated and cut. A sublaryngeal tracheotomy is performed, carefully avoiding the cutting of vessels, and a conic cannula is introduced. The abdominal aorta and inferior vena cava are exposed, ligated and metallic cannulas with threaded surface introduced proximally, and the vessels ligated over them. Rubber tubes lead off from the four openings and all incisions are sutured. Ice tongs or toothed retractors are used to hold the skull and the body is then suspended vertically on a rope or preferably on a swivel joint in front of the fluoroscopic (radiographic) table, with the feet just touching the floor. The lungs are inflated under fluoroscopic control, avoiding displacing the diaphragm too far caudad. Acetate of lead or a fairly thick solution of barium sulphate, to which pulverized red oxide of lead may be added, are used as contrast media. The injection is made slowly, with a syringe holding 150 cm<sup>3</sup>. About 750-1500 cm<sup>3</sup>. of contrast fluid are needed to fill one or the other side (cardiac cavities plus respective vessels). One proceeds to inject either the right cavities and veins through the cannula in the jugular vein or the left cavities and arteries through the catheter in the carotid artery. Roentgenograms are taken, with a target-film distance of 200 cm. or more, preferably in the anterior, left lateral and both anterior oblique views. The degree of rotation should be carefully noted. This being done, one proceeds to open the proper abdominal tube. A part of the contrast medium escapes, much of it is rinsed out by slowly injecting water into the proper neck tube. Contrast medium always remains in the pulmonary artery and its branches with injection of the right side, in the left atrium and pulmonary veins when the left side has been injected. Occasionally the ventricles cannot be emptied completely. Other roentgenograms may now be taken to determine the amount and location of the remnant of contrast material. The injection of the contralateral cavities and the respective vessels follows and roentgenograms are taken again, for the oblique views. super-imposed drawings of the cavities to each other is investigated. selective

The following difficulties may be encountered. The catheter may deviate into the descending aorta or, having been pushed too far in the correct direction, may penetrate the ventricular wall. Clots in the cardiac cavities and pulmonary artery may interfere with complete filling. The presence of a pleural effusion interferes with good visualization, this is overcome, when a laparotomy is done, by making a small opening with the finger in the lateral-caudal portion of the diaphragm; drainage into the abdominal cavity results as soon as inflation of the lungs is begun.

Postmortem injection experiments in the new-born give results different from those obtained in the child or adult. The reason for this is the presence of two fistulas, one within the heart (foramen ovale) and one between the aorta and pulmonary artery (ductus arteriosus). An injection of contrast medium into the jugular vein or inferior vena cava will opacify both vv. cavae, the whole venous circulation, both atria, both ventricles, aorta and pulmonary artery. An injection of contrast medium into a carotid artery or into the abdominal aorta will fill both arterial systems including the coronary arteries;

the aortic and pulmonic leaflets will prevent an appreciable flow of contrast medium into the left and right ventricles.

A critical attitude is necessary as far as deductions drawn for the anatomy of the living are concerned. In the cadaver the diaphragm is in a high position and the chest is in the expiratory phase. Death, postmortem changes, formalin fixation, freezing and injection experiments, all occur and are made as a rule in the horizontal position, whereas most roentgen ray studies of the chest in the living are made with the subject in the upright position.

As to the application of the roentgenologic method to the postmortem study of the blood vessels in health and disease, and especially of the coronary arteries, the reader is referred to the bibliography of this chapter and to chapter I.

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### CHAPTER III

NORMAL CARDIOVASCULAR SYSTEM, STATIC  
INTRATHORACIC

From the viewpoint of radiation physics, the intrathoracic cardiovascular system presents, under normal conditions, a rather homogeneous mass with the exception of transparency due to air in the tracheo-bronchial system; diagnosis is, therefore, made mainly by contours. Those contours are visible which are coated by the mediastinal pleura, provided normal adjacent lung tissue is present. The inner topography is not directly observed. The border of the silhouette shows subdivisions either curved or almost straight and of very similar appearance for different silhouettes.

The following descriptions should be read together with simultaneous observation of roentgenograms.

[illegible]

diaphragm-liver silhouette may be bridged by a short shadow formation. Its anatomical basis is not constant; it may correspond to the hepatic veins or to a pleuro-pericardial fold with an extra-pericardial fat pad. *Left Side.* Commencing with the cranial end, at the clavicle, a short vertical or oblique border is represented by the left subclavian artery or vein. This is followed by a rather short, semicircular or oval curve; it depicts the fusion of the dorsal portion of the aortic arch and the descending aorta; its caudal continuation disappears, more or less obliquely, within the total shadow and is overlapped by a second rather flat arch. The junction of these two arches may be seen to be spanned by a short straight contour, the edge of the ligamentum arteriosum. The flat arch indicates the pulmonary artery in the cranial portion and in the caudal portion the uppermost part of the right ventricle (conus arterialis), however, recognized only by the left auricular appendage. The well-curved, elliptical border, back toward the midline near the heart, is the arch of the aorta. This arch, the longest of

all, corresponds to the left ventricle. The left cardio-diaphragmatic angle is often spanned by a triangular, less dense shadow. This formation is caused by a fold which extends from the pericardial surface to both mediastinal and diaphragmatic pleurae and, at its ventral surface, it is usually covered with fat tissue.

The semicircular or oval aortic contour at the cranial aspect of the left side is absent or only very shallow in children. It frequently reaches as high as the level of the left clavicle. A slightly better visualization is obtained in recumbency.

**TOPOGRAPHY, VISIBLE.** The convex contour of the distal part of the ascending aorta is sometimes visible just inside the shadow of the superior vena cava. The cranial contour of the aortic knob may be followed mesially until it crosses into the denser shadow of the spine. The lateral contour of the descending aorta

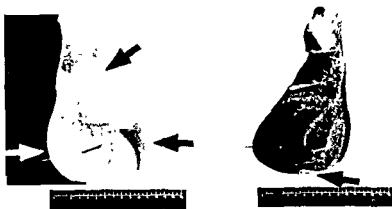


FIG 5—Young male, healthy. Normal heart. Roentgenologic, volumetric reconstruction. *Ant view:* rt atrium (rt arrow) Pulmonary artery (lt upper arrow) Lt ventricle (lt lower arrow) *Post view:* rt ventricle (arrow)  
Courtesy Prof Dr G G Palmieri, Bologna, Italy

is partially visualized through the pulmonic and cardiac shadow. The transparent lumina of the trachea and the main bronchi are visualized by means of adequate technique.

The pericardium has been omitted in this description, it would be more correct to speak of "pericardial surface" instead of "cardiac surface."

**TOPOGRAPHY, NOT VISIBLE, STUDIED FROM POSTMORTEM EXPERIMENTS.**  
1) **Vascular area.** To the right and dorsad to the ascending aorta is the band-like superior vena cava. It overlaps the ascending aorta in the young but is overlapped by it in old age, aortic disease, and perhaps occasionally in the healthy adult. Its upper limit is usually at the level of the 7th (occasionally 8th and even 9th) thoracic vertebra. The shape of the ascending aorta is fusiform; its origin is above the center of the cardiac area, therefore to the left of the midline, at about the level of the 8th (7th to 9th) thoracic vertebra. The arch of the aorta in the young hardly reaches the level of the 4th thoracic segment and has an antero-posterior course, with the brachio-cephalic vessels situated more in juxtaposition. The ascending and descending aorta may represent a complete spread in the aged. The ascending aorta is partially overlapped by the pulmonary conus and artery. The latter of

itself has a marked antero-posterior course and is therefore foreshortened. Its bifurcation corresponds to the middle or upper third of the vascular pedicle. The main right branch passes below the bifurcation of the trachea. Its caudal border corresponds about to the lower limit of the superior vena cava. The main left branch passes behind the trunk of the pulmonary artery. 2) Cardiac area. A rather small, bandlike area along the left contour corresponds to the anterior wall of the left ventricle. The interventricular sulcus as seen from without is a misleading landmark for the inner topography since the interventricular

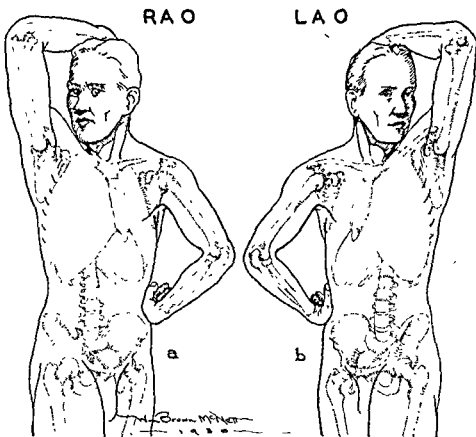


FIG. 6—Positions for anterior oblique views. Degree of rotation to be determined for each individual by means of fluoroscopy a—rt ant obl view b—lt ant obl view.

septum is curved medially thereby causing the left and right ventricular cavities to overlap considerably in projection. Thus the medial border of the left ventricle is convex, similar to its lateral (left) border. The left ventricle forms the lower pole proper. The ventral wall of the right ventricle takes up the central area, it is wider caudally and smaller cranially. The right ventricular cavity has two portions. One is horizontally situated, triangular in shape and communicates at its base with the right atrium, while its lower contour forms the lower cardiac border; this is the inflow tract. Grafted on it and situated vertically is the outflow tract, the infundibulum; its left border is much

longer than the right and reaches approximately to the level of the intersection of the left middle and lower contour of the silhouette (point G) (Fig. 31). The right ventricular lower pole portion is, as a rule, 0.5-3 cm. craniad to the corresponding portion of the left ventricle and this is always the case with the vertical type of heart. The infundibulum and the top portion of the left ventricle join near the aforementioned point G.

The caudal heart border is visible in the presence of much gas in the stomach, an interposition of the gas-containing colon between the diaphragm and the liver; with a pneumoperitoneum when in the upright position; and with a pneumopericardium when in the Trendelenburg position. The right atrium always forms the right heart border and occupies the right and right upper area of the ventral surface. Its medial border may reach as far as the vertebral axis. The left atrium is superimposed in projection over the inflow tract of the right ventricle and over the greater part of the right atrium; it extends farther craniad than the latter. Postmortem injections never show it as reaching the

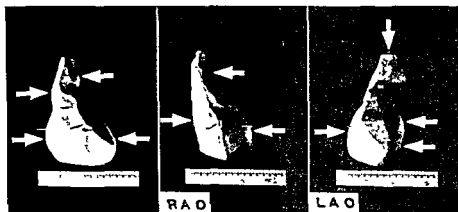


Fig 7—Young female, healthy. Normal heart. Roentgenologic, volumetric reconstruction. Ant view: rt atrium (rt lower arrow). Cranial portion, ascending aorta (rt upper arrow). Distal portion aortic arch (lt upper arrow). Lt ventricle (lt lower arrow). Rt ant obl view: normal, almost straight dorsal border, lt atrium (rt arrow). Ventral contour cranial portion ascending aorta (lt upper arrow). Rt ventricle (lt lower arrow). Lt ant obl view: rt ventricle (rt arrow). Vessels to neck (upper arrow). Lt atrium (lt upper arrow). Lt ventricle (lt lower arrow). Courtesy Prof Dr G G Palmieri, Bologna, Italy.

left silhouette contour but it is probable that in the living, during certain phases of the cardiac cycle, the left auricular appendage does form a small portion of the border.

The aortic valves are situated approximately craniad to the center of the cardiac area and somewhat to the left of the midline. Slightly craniad and to their left are the pulmonary valves. The tricuspid valves are caudad and in the midline. Slightly craniad and to their left are the mitral valves. Variations, depending upon the shape and the position of the heart, must be considered.

The axis of the inferior vena cava corresponds in projection to the right sternal border.

The use of the term "apex of the heart" has been avoided. The term "lower

pole" which has been substituted is preferable but still far from correct. It is obviously impossible to determine in any of the projections a 'localized "apex" or even the general direction of this apex or lower pole. Furthermore, it is impossible to state whether this left lower pole points ventrally or dorsally unless the heart is actually reconstructed as a three-dimensional model.

It is also necessary to emphasize that the left lower pole region does not give a correct concept of the caudal extension of the heart as may be shown by adhering to the following routine. The junction of the left lower contour with the diaphragm may be marked orthodiagraphically. The patient is then rotated 50° into the left anterior oblique position and the same procedure is repeated for the most caudal point of the left lower contour. It is found



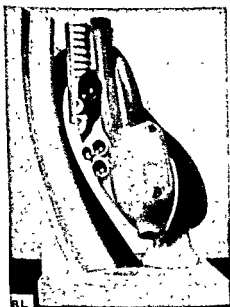
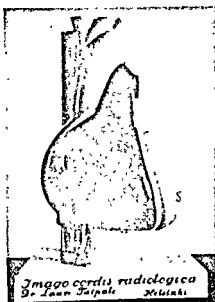
FIG 8—Anatomic specimens were hardened in situ and sketches obtained by means of an orthogonal projection. Case 1, upper row, died from postnatal hemorrhage (dash line—border of thymus); case 2, middle row, from bilateral pneumonia; case 3, lower row, from tuberculous meningitis. 0°—ant view, 45°—lt ant. obl view, 90°—lt lat view, 315°—rt ant. obl view. / / / /—rt. heart cavities, \ \ \ \—lt. heart cavities; respective atria darker. From Dietlen, H and Schall, L in *Handb Röntgendiagn u Therapie Kindesalter*, ed by Engel, St., Schall, L., 1933, p. 400-401. Courtesy Prof. Dr. H. Dietlen, Homburg, Saar and G. Thieme, Leipzig, publisher.

that in  $\frac{2}{3}$  of all cases this latter level is from 0.5 to 2.0 cm more caudad. It

which continues into a straight contour. This may or may not represent the ventral outline of the ascending aorta, for postmortem opacification experiments have shown that ventrad to the aorta there may lie a bandlike area of the pedicle, several mm. in width and apparently not formed by the aortic

wall. Further study on this problem is needed. The next arch is oblique, long, slightly curved and finally makes a rather sharp turn inward just above the diaphragm. Unless the degree of rotation is very slight the right ventricle

FIGS 9, 10, 11—Dr. Lauri Taipale's model, photographed in the ant, rt lat and lt lat view. It was reconstructed on the basis of many roentgen observations on healthy individuals. It is highly recommended for the study of the normal anatomy, particularly for the roentgenological approach. This model may be ordered from A. B. Christian Nissen OY., Centralgatan 3, Helsingfors, Finland.



alone participates in the contour. *Dorsal Aspect* Approximately at 30-35° the mediastinal mass becomes separate from the spinal shadow. The size of the transparent retrovascular and retrocardiac spaces is increased by further rotation and during inspiration. At a certain angle, a few cm. ventrad to and





FIG 16

Figs 12, 13, 14, 15, 16—50 yrs, M Healthy No complaints  
Normal findings. *Ant view* slight degree of elongation of aorta, perhaps with paravertebral type of descending aorta. *Rt side* contour of distal portion of ascending aorta (upper wh arrow), contour of rt atrium (lower wh arrow) *Lt. side* aortic knob (upper wh arrow), contour of descending aorta (middle wh arrow), contour of lt. ventricle (lower wh arrow), mesial contour of arch of aorta (upper bl arrow), contour of pulmonary artery (lower bl arrow). *Rt ant obl views* (A, B and C taken at shorter distance, B with lesser, C with high degree of rotation) ventrally: ventral contour of distal portion of ascending aorta (upper wh arrow), division of pulmonary artery (lower wh. arrow), ventral contour of trachea (middle wh short arrow); conus arteriosus (bl arrow) Dorsally: contour of sup vena cava (middle wh arrow), ventral contour of descending thoracic aorta (lower wh arrow), dorsal contour of trachea (upper short wh arrow); contour of lt. atrium (upper bl arrow), contour of rt atrium (lower bl arrow). *Lt. ant. obl. view*: ventrally: contour of rt ventricle (lower wh. arrow), ventral contour of trachea (upper short wh arrow). contour of lt. atrium (middle bl arrow); contour of lt ventricle (vertical wh arrow) *Figs 12, 13, 16* from Roesler, H. in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System, ed. by Ross Golden, 1936, p 202-204 Courtesy Th Nelson & Sons, New York.

FIG 13



FIG 15

FIG 12

FIG 14

Dorsally dorsal contour of descending thoracic aorta (upper bl arrow), contour of lt atrium (middle bl arrow), contour of lt ventricle (lower bl arrow), dorsal contour of trachea (upper short wh arrow), main lt branch of pulmonary artery crossing through aortic window (vertical wh arrow) *Figs 12, 13, 16* from Roesler, H. in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System, ed. by Ross Golden, 1936, p 202-204 Courtesy Th Nelson & Sons, New York.

parallel with the spine, a straight contour is noticed which is the ventral aspect of the descending aorta. It is best seen within the lower part of the retrocardiac space and becomes less and less distinct craniad. Here the transparent band of the trachea and the left main bronchus is clearly visible and its ventral edge delineates the silhouette at this level. However, farther dorsad a new faint border may become visible, provided the rotation is increased. It can be traced through the transparent band of trachea and bronchus and is the dorsal border of the superior vena cava. Farther caudad, and likewise at a higher degree of rotation, follows a small, rounded prominence, representing the dorsal aspect of the right pulmonary branch. Finally, a rather flat arch is noted which increases in its curvature at its lower aspect. This silhouette corresponds to the left atrium above and the right atrium below. At a lesser degree of rotation the greater part is formed by the right atrium. The cardio-hepatic angle is bridged by the shadow of the inferior vena cava.

**TOPOGRAPHY, VISIBLE.** When the rotation proceeds to approximately  $35^\circ$  the aortic shadow has a club-shaped or racquet-like appearance, with the wider end craniad. This is caused by the oblique course of the left bronchus, the air content of which almost completely extinguishes the shadow of the rest of the aorta. At a rotation of  $40-50^\circ$  the courses of the bronchus and of the ventral contour of the ascending aorta are almost parallel and here the aortic shadow assumes a band-like appearance. The cranial contour of the pulmonic arch in front may be seen to curve through the aortic shadow. In



FIG 17—53 yrs, M Duodenal ulcer Normal cardiovascular system Lt lower thoracic region Ant view. Lt lower cardiac contour just visualized (bl arrows). A triangular shadow extends beyond it, with slightly curved lat contour (wh arrows). Marked development of extrapericardial pleural veil and fat tissue. This shadow formation should not be included when roentgenological cardiac measurement is taken.

front of the left bronchus and slightly caudad of the level of the pulmonic arch a darker, round area is noted. This corresponds to the cross section of the left main branch of the pulmonary artery. Haziness and mottling are present below the bifurcation, and dorsal to the contour of the left atrium. The pulmonic veins as well as the projection of the right hilus into the retrocardiac space account for this.

The visibility of the aortic arch, dorsad to the trachea, increases with age; this is due primarily to the change in its course.

**TOPOGRAPHY, NOT VISIBLE, STUDIED FROM POSTMORTEM EXPERIMENTS**

1) Vascular area. The arch of the aorta is much foreshortened, especially in



above down, with the aortic and mitral valves near the center of the cardiac area. The left coronary artery shows a long curved arch consisting of the main branch and the anterior descending branch that curves just inside of the cranial-ventral silhouette contour. The right coronary artery courses obliquely caudad-dorsad toward the diaphragm.

**Left Anterior Oblique View. SILHOUETTE. Ventral Aspect.** Like the anterior view the cranial contour is composed of the innominate vein and



FIGS 20, 21.—74 yrs F Aortic and cardiac enlargement Normal B P Death from cerebral hemorrhage Postmortem opacification *Lt ant obl views* A contrast medium was injected through the rt internal jugular vein and a roentgen film was taken The barium was then rinsed out from both venae cavae and from the rt heart cavities B contrast medium was injected through a catheter which had been pushed from the rt common carotid artery into the lt ventricular cavity Barium is noted to persist, from the previous injection, in the pulmonary artery and in its branches A rt branch pulmonary artery (rt upper bl arrow) Lt branch pulmonary artery (lt upper bl arrow) Pulmonary artery (lt bl short arrow) Outflow tract rt ventricle, conus arteriosus (rt bl short arrow) Sup. and inf vena cavae, entering rt atrium (upper and lower short bl arrows) Inflow tract rt ventricle (lower bl arrow) Coronary vein (wh arrow) B arch of aorta (upper bl arrow) Ascending aorta, first portion (rt upper bl arrow) Descending abdominal aorta (rt, lower bl arrow) Lt ventricle (wh arrow) From Laubry, Ch, Cottenot, P, Routier, D, Heim de Balsac, R *Presse mtd*, 1935, Dec 21, fig 10 and 11 Courtesy Dr R Heim de Balsac and Masson & Cie, publisher, Paris

the superior vena cava Caudally there follows a shorter convex arch, the ventral aspect of the ascending aorta This contour changes direction with a flat angle and below the junction, the silhouette is made up either entirely by the right ventricle (at a higher degree of rotation) or (at a lesser degree) in its cranial part by the right auricular appendage, and in its caudal aspect by the right ventricle **Cranial Aspect** The left border of the brachiocephalic shadow corresponds to the left carotid artery. Dorsad to it, the curved outline of the cranial border of the aortic arch is visible and may be followed into the

dorsal contour of the descending aorta. *Dorsal Aspect.* At approximately  $40^\circ$  the cardiac mass becomes separated from the vertebral shadow. The cranial contour begins rather indefinitely in the continuation of the left main bronchus. It is first directed obliquely toward the spine and farther caudad deviates from it to fuse with the outline of the left diaphragm. The cranial portion of this contour outlines the left atrium, the caudal one the left ventricle.



FIG 22—Contrast visualization by means of intravenous injection of 70% diodrast Lt ant obl view. Note the cavity of the rt ventricle (1), its curved dorsal outline (2) corresponds to the interventricular septum, the thickness of the wall of the rt ventricle (3) can be measured ventrally. The pulmonary artery (4), ascending to its bifurcation, the lt main branch (5), and the smaller branches are clearly outlined. Courtesy Drs G P Robb and I Steinberg, New York City, NY



FIG 23—Contrast visualization by means of intravenous injection of 70% diodrast Lt ant obl view. Note the cavity of the lt ventricle (1), its curved ventral outline (2) corresponds to the interventricular septum, the thickness of the wall of the lt ventricle (3) may be measured dorsally. The ascending portion of the aorta (4) is covered in front by the shadow of the sup vena cava. Courtesy Drs G P Robb and I Steinberg, New York City, NY

*Caudal Aspect.* The heart border may show near its fusion with the abdominal shadow a shallow indentation which is better observed while studying the pulsations. This indicates the region of the interventricular sulcus.

**TOPOGRAPHY, VISIBLE** When the patient is sufficiently turned ( $45^\circ$ ), a shadow is seen coursing vertically through the outline of the ascending aorta. It coincides nearly with the termination of the transparent right bronchus and represents the ventral border of the superior vena cava. The area of the bifurcation contrasts brightly and is known as the aortic window. A curved handlike density is seen crossing it which represents the left main branch of the pulmonary artery. A translucent area is seen surmounting the aortic arch. Its right border is formed by the left subclavian artery, the left border is formed by the spine, and its base by the arch of the aorta. This is referred to as the "aortic triangle."

## TOPOGRAPHY, NOT VISIBLE; STUDIED FROM POSTMORTEM EXPERIMENTS.

1) Vascular area. The inner contour of the aorta is nowhere visible with the possible exception of the very short distance when it crosses the lumen of the right bronchus and then only at certain different angles. The inner border closely follows the cranial angle between trachea and left bronchus. The origin of the ascending aorta descends deep into the center of the cardiac silhouette and the direction of its axis is from ventral-cranial toward dorsal-caudal. The lumen of the superior vena cava overlaps the lumen of the ascending aorta to varying degrees. This depends upon the degree of rotation. Beyond 40° or 45° its ventral contour recedes behind the corresponding aortic contour. The pulmonary artery, together with its two branches, forms a T-like figure, with the right branch superimposed on the dorsal ascending aortic contour.

2) Cardiac area. The cranial-dorsal aspect is occupied by the left atrium; the caudal-dorsal aspect by the left ventricle. Both ventricular cavities partly overlap each other. The ventral aspect is taken up by the superimposed right ventricle and atrium. The course of the interventricular sulcus is not well known; it is probably near the center of the cardiac shadow at an angle of rotation lying between 45° and 55°. The interventricular septum is curved in a spiral manner, thus a linear projection cannot be obtained. The valve areas, pulmonic, aortic and tricuspid from above down, can be placed on a line which runs from craniad-dorsad to caudad-ventrad, with the mitral valves farther dorsad and between the levels of the aortic and tricuspid valves. The anterior descending branch of the left coronary artery is foreshortened and descends caudad, the circumflex branch courses horizontally toward the spine. The right coronary artery curves in a quarter circle from horizontal to caudad toward the diaphragm and lies near the ventral silhouette border.

**Lateral View. SILHOUETTE. Ventral Aspect.** The arch sequence is as follows: cranial part of the ascending aorta, pulmonary artery and conus arteriosus, and right ventricle. The latter contour fuses with the sternal shadow in front of it. **Dorsal Aspect.** The area of the left atrium is above and that of the left ventricle below. Caudally the inferior vena cava bridges the cardio-hepatic angle. In the supine position the level of the dorsal border of the cardiac shadow is from 6.5-10 cm. ventral to the skin of the back. This distance represents 35-50% of the depth diameter of the chest. To know this distance is of practical importance when the venous pressure is determined by the direct method. The point of measurement—usually the cubital fossa—should be placed at the level of the right atrium.

There is very little contrast within the silhouette with the exception of the tracheobronchial transparency.

## TOPOGRAPHY, NOT VISIBLE; STUDIED FROM POSTMORTEM EXPERIMENTS.

1) Vascular area. The ascending aorta is more convex in older people. The ventral contour of the ascending aorta and the cranial contour of the pulmonary artery cross approximately 1 cm. from the former's origin. The pulmonary artery is overlapped by the ascending aorta and superior vena cava. The latter is situated half-way between the sternum and the spine.

2) Cardiac area. There is a maximum of overlapping of both ventricular and atrial cavities, respectively, with the right ventricle slightly surpassing dorsad. Three valve areas form the corners of a triangle, the pulmonic craniad and

ventrad, the tricuspid caudad and ventrad, the mitral dorsad and more caudad, with the aortic valve area situated within the cranial portion of the triangle.

**Elements.** It is often necessary to analyze one of the single elements, the whole of which make up the total cardiovascular shadow. Each of these elements may be studied directly, i.e., by a tangential view; and indirectly, by analyzing those changes which occur in the size, shape and position of the other elements, adjacent or remote. Certain views (positions) are preferable for certain information. Standardized angles cannot be given for the oblique views. The patient should be rotated under fluoroscopic guidance until the most information is obtained. Unless otherwise stated, the patient is always con-

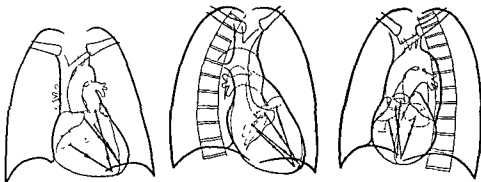


FIG 24—Schematic drawing to indicate the concept of the inflow and outflow tracts in the rt. and lt ventricles. The heavily drawn lines are roentgenologically visible while the thinly drawn and dashed lines serve for an understanding of the topography of the surface and deep structures of the heart and great vessels. The arrows indicate the course of blood flow. The views are from lt to rt Ant, rt ant obl and lt ant obl.

In a personal communication, Isaac Starr, M.D., pointed out that the physiological lines of importance would run from the valves, not to the apex, but to the center of mass of the blood in the ventricles of the heart. Courtesy Doz. Dr. E. Zdansky, Vienna, and J. Springer, publisher, Vienna. From *Röntgendiagnostik des Herzens und der grossen Gefässe*, 1939, Fig. 62.

sidered as being started from the standard position, namely, the anterior view. For the study of the single elements of the cardiovascular shadow the following views should be chosen:

**Left Ventricle.** 1) rotation counter-clockwise into the right anterior oblique view, for approximately  $20^\circ$ . 2) rotation clockwise through the left lateral into the right posterior oblique view, for  $210^\circ$ .

**Right ventricle.** 1) rotation counter-clockwise into the right lateral view, of  $90^\circ$ . 2) rotation from the left anterior oblique into the left lateral view, for  $60^\circ$ .

**Left atrium.** 1) rotation from the right anterior oblique through the right lateral into the right posterior oblique view, for roughly  $90^\circ$ . 2) rotation from the left anterior through the left lateral into the left posterior oblique view of about  $90^\circ$ .

**Right atrium.** 1) rotation counter-clockwise through the right lateral and posterior into the left posterior oblique view, for approximately  $190^\circ$ . 2) rotation clockwise into the left anterior oblique view, of  $30^\circ$ .

**Ascending aorta, distal portion.** 1) rotation counter-clockwise into the right

lateral view, for  $90^\circ$ . 2) rotation clockwise into the left lateral view, for  $90^\circ$ .

Arch of the aorta. Rotation clockwise into the left lateral view, to  $90^\circ$ .

Descending aorta, cranial portion. 1) rotation from the left anterior oblique into the left lateral view, for  $45^\circ$ . 2) rotation from the posterior into the right posterior oblique view, of  $15^\circ$ .

Descending aorta, caudal portion. Rotation from the right anterior oblique into the right lateral view, for roughly  $30^\circ$ .

Pulmonary artery. 1) rotation counter-clockwise into the right anterior oblique view, to  $60^\circ$ . 2) rotation clockwise for  $30^\circ$ .

Superior vena cava. Rotation counter-clockwise into the right anterior oblique view, of  $70^\circ$ .

Inferior vena cava. Rotation from the right anterior oblique into the right lateral view, for approximately  $70^\circ$ .

**Constitutional Features** The general contour of the chest cavity determines the form and position of all the organs contained. It has been demonstrated by anatomic sections that the outline of the circumference of the heart is in close relationship to the form of the circumference of the chest. Hearts which are located in round chests have a more circular circumference, while those in flatter chests are more oval. Furthermore, in those which are transversely placed in a broad chest, the right atrium, right and left ventricle are in contact with the anterior chest wall, while hearts situated vertically in a narrow chest show practically only the right ventricle in contact, with the right atrium situated farther dorsad and cranial. Only in rare instances does the right ventricle form the caudal portion of the right border of the heart. In a narrow chest, the arch of the aorta displays a more sagittal course, while the origins of aorta and pulmonary artery are closer together. In a flat chest, the course of the aortic arch is decidedly more frontal and the origins of the two large vessels are farther apart. The translation of such findings into the language of roentgenology is indeed simple.

Individuals in whom the development of the extremities and the vertical dimension prevails over the width development, trunk volume and visceral mass and who show in addition a rather frail bone and muscular development, usually give the following roentgenologic appearance. The mediastinal shadow as a whole is narrow but long. The diaphragm is low in position, the 11th rib being frequently visible above its right dome, its inspiratory excursion is relatively limited. The convexity of all contours of the silhouette, in all views, is only slightly prominent, however, the pulmonic arch is relatively prominent. There is little angulation between the main axes of the vessel and heart shadow, respectively. The cardiac silhouette is more centrally located, appears pear-shaped and its area of contact with the abdominal shadow is relatively small. The caudal aspect of the right border may show a shallow intersection which is usually determined only by fluoroscopy. Most probably the right ventricle participates in the right heart border below this junction and above the cardio-diaphragmatic angle. The vertical dimension of the vascular shadow is considerable. The arch of the aorta appears short in the left anterior oblique view and is curved on a small radius. The lateral view reveals a diminished inclination of the mediastinum; both the dorsal and ventral cardiac silhouette contours are rather flat and the latter is



often completely visible. The retrocardiac space is comparatively small. Extreme types of hearts as here outlined are known as *cor pendulum* and *drop heart*. As to certain aspects in the functional evaluation of the vertical, small heart, see chapter VII.

Individuals in whom the development of trunk and width prevail over length and extremity, and who show a marked development of viscera and muscles reveal a different roentgenological picture. The mediastinal shadow as

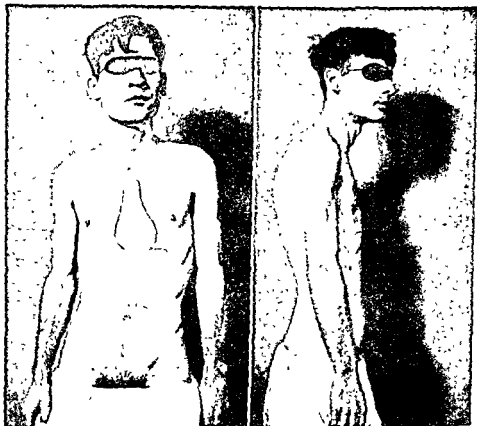


FIG. 25.—23 yrs, M. Schizophrenic. Hyposthenic constitution. Height 170 cm. Weight 57 kg. Normal clinical cardiovascular findings. L 12.0, B 9.0, T 9.4, Th 25.3 cm. Vertical type of heart with relatively low position of diaphragm. When T is used exclusively, the size of heart is underestimated with this constitutional type.

a whole is short and wide. The diaphragm is high in position, the 9th rib or possibly only the 8th rib is visible above the right dome; its expiratory excursion is diminished. The convexity of all contours of the silhouette is marked and noted in all the views. There is a sharp angulation between the main axes of the vessel and heart shadows, respectively. The heart area appears decreased in height, is boot shaped, is situated predominantly to the left, and fuses with the abdominal shadow to a considerable degree. The vertical dimension of the vascular shadow is diminished. In the left anterior oblique view the arch of the aorta is curved on a large radius. The lateral view reveals an increased

inclination of the mediastinum, both the dorsal and ventral cardiac silhouette contours are sharply curved and the latter fuses broadly with the sternal shadow. The retrocardiac space is increased. This type is indicated as horizontal or transverse.

**Age.** The cardiovascular shadow in early childhood reveals less differentiation than in the adult. The formation of well defined arches and their junctions has not yet occurred. The appearance in the newborn is often almost entirely globular. This is due in part to the superimposition of the thymus

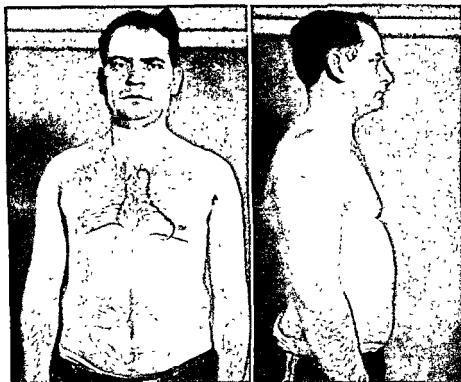


FIG 26 — 28 yrs, M healthy, no complaints. No history of athletic activity. Hypersthenic constitution. Height 173 cm, weight 77 kg, chest circumference 96 cm. Normal clinical cardiovascular findings. L 12 1, B 8 9, T 11 8, Th 25 4 cm. Oblique type of heart with relatively high position of diaphragm.

shadow, and also to the fact that the width of the heart is considerable as compared to its length. The atria and auriculae are comparatively large, as is also the right ventricle. Variations in shape can be accounted for, mainly, by the varying appearance of the thymic shadow. After it has regressed, the vascular shadow appears short and relatively wide. The right cardiac border is comparatively long, and markedly curved, which feature may continue for several years. There is no aortic knob visible and the pulmonic arch is not prominent except during a crying spell. The caudal heart border is visible to a large extent and occasionally entirely so. The transverse position of the heart begins to change slowly, into an oblique position, between the 6th and 12th

month. The unequal speed of growth between the lungs and the chest wall, and between the chest wall and the spine, respectively, leads to *this descent of the heart*. A more or less definite shape of the cardiovascular shadow is rather well established between the sixth and eighth year. A typical drop heart is very rarely met with but otherwise different constitutional types are observed. *The right border of the vascular shadow is always rather straight and this indicates that the superior vena cava forms this contour*. The short and plump appearance of the vascular pedicle and the relative fullness of the waistline of the silhouette, as frequently observed in children, is due in part to special conditions of projection. In the adult the width of the chest much prevails over its depth; and the angle of inclination of the heart, as seen in the lateral view, is rather steep. When the chest is relatively small but deep and the diaphragm rather high, such as noted in childhood, heart and great vessels hold a somewhat different position: as seen in the lateral view, the great vessels ascend dorsad rather obliquely and the inclination of the heart is less outspoken. Hence, in the anterior view, heart and great vessels reveal a projective foreshortening, i e., they are visualized somewhat from below. The vascular pedicle is short, plump, and the waistline is full. If the chest is now changed from an erect to a somewhat bent-forward position, the fullness of the waistline will be noted to diminish promptly. Such a situation incidentally holds true not only for children but is also occasionally noted in pregnant women, the reason being the same.

*The cardiovascular shadow in the aged shows the vascular shadow comparatively long and wide. The right cranial border is rather prominent and convex and the vertical distance from the right cardiovascular junction to the level of the cranial contour of the aortic arch is considerably elongated, absolutely as well as relatively, when compared with the height of the heart. It is probable that the ascending aorta at this level actually represents the mediastinal border. The aortic knob projects rather far craniad and laterad. The left lateral contour of the descending aorta is visible to a considerable length in its almost vertical caudal course and it may intersect the cardiac shadow in the region of the left auricular appendage. The cardiac silhouette often reveals a trans-*

*Calcification is*  
*ht anterior*  
*ormally its*  
*axis converges craniad with the axis of the spine. Here it diverges and its course is toward the ventral chest wall. The aortic arch becomes visible; below and enclosed by it a transparent area is seen, representing a small aortic window.*  
*throughout*  
*the aorta*  
*ig contour*  
 and a large aortic window. The cranial arch contour may be visualized through the tracheal transparency.

**Relations.** The relation of the tracheobronchial tree, that of the esophagus and of the diaphragm to the cardiovascular system is of considerable interest and importance in cardiovascular pathology and, therefore, the normal features should be known.

**TRACHEA.** *The trachea is seen to be in close relationship to that part of*

the arch where its proximal-ventral portion changes its oblique course into the distal-dorsal portion which has an almost sagittal direction. Its transparent band interferes with attempts to measure correctly the cephalic part of the ascending aorta, in the right anterior oblique view. The elongated and widened vessel in the aged causes a fairly marked impression on the trachea.

**ESOPHAGUS.** Its lumen is demonstrated by means of barium paste. At the level of the arch one sees a curve in the barium stream; this is designated as the aortic arch impression. The curve is shallow and flat when the deviation commences below the summit of the arch. Here the esophagus is in close relationship to the very top portion of the descending limb. In other instances the curve is rather deep, delineating particularly well a part of the caudal aortic circumference. The convexity of the curve is directed to the left in both the anterior and right anterior oblique views. Caudad to this curve, in the right anterior oblique view, one sees another deviation, rather short and flat in type and sometimes hardly visible. This curve is at the level of the right main pulmonary artery branch but is separated from it by the left main bronchus. The farther course of the esophagus reveals a curve of slightest degree at the level of the left atrium.

Its concavity is to the left and ventrad. It is liable to be absent in subjects of tall stature and during forced inspiration, while it is better visualized in subjects of short stature, in children and during forced expiration. The esophagus crosses the lowest third of the descending aorta with a slight concavity directed to the left. With marked elongation of this vessel in older people a more marked deviation of the barium stream is noted to the left in the anterior view and dorsad in the left anterior oblique view.

**DIAPHRAGM.** Oblique and lateral views show that the diaphragm descends from its dome dorsally and ventrally and the cardiac shadow completely occupies, in front, the central portions of the costo-diaphragmatic sinus. In the left anterior oblique view both leaves of the diaphragm are seen to form an angle. At a rotation of  $48^{\circ}$  to  $55^{\circ}$  the dorsal-caudal lower contour intersects with this angle; in the presence of a globular or transverse type of heart this angle may be slightly overlapped by the cardiac shadow. With a vertical small heart the angle is located between the cardiac shadow and the spine.

**Influences. BODY POSITION.** The examination of the body in different positions shows changes in the size, shape and position of the cardiovascular sil-



FIG 27—78 yrs, F *Rt ant obl view*—calcification of cartilaginous rings in trachea and major bronchi. Bend in course of trachea at level of aortic arch (ant bl arrow). Dorsal contour of sup vena cava (post bl arrow)

houette. We are inclined to think too much, perhaps, of marked actual changes in form and volume. Thus, the dissection of the hearts of dogs whose bodies were frozen in different positions shows but little gravity influence on the form of the heart. We judge changes in position by taking, for instance, the ribs as landmarks. Here we are dealing with an elastic system subject to many forces. Thus, with a change from the upright to the recumbent position, the sternum moves caudad and dorsad, the ribs following. In the lateral prone positions, the ribs of the descending side move caudad and the depth on this side is increased. The shifts of organs observed are often only apparent.

The change from the recumbent to the vertical position decreases the angulation and narrows and lengthens the vascular shadow. The area and the transverse diameter tend to decrease, especially so if the heart rate increases considerably, and the oblique diameter increases. Measurements in the lateral view have shown in a few cases that the depth diameter decreases as well. It is certain that actual volumetric changes take place.

In the recumbent-lateral positions the lower leaf of the diaphragm ascends, and increases its respiratory excursions, while the upper one descends. The whole mediastinal shadow sinks into the dependent side and the cardiac silhouette changes in outline. The mediastinal shadow returns on deep inspiration, against gravity, toward the midline. The caudal heart border is considerably influenced by the adjacent, elevated diaphragm and it is advisable to study, preferably, the movements of the cranial border of the cardiac silhouette. The degree of lateral displacement varies from 1.5-3.0 cm. but may be as high as 5.0 cm. in asthenics, in persons who lost weight rapidly, or in women with poor abdominal muscle tone subsequent to many deliveries or to a very recent delivery. Generally a thin walled and poorly filled heart changes more readily in position than does a thicker walled and well filled heart. A prerequisite for testing out for lateral displacement is the patient's ability to breathe deeply.

**RESPIRATION** Relatively slight changes are noted during quiet respiration.

Forced inspiration, with the costal type of respiration prevailing, shows no vertical shift or only a slight rise. With a prevailing diaphragmatic type of respiration, a caudal displacement is noted. In addition to the cranio-caudal component, a lateral-medial one is to be considered. One observes the left heart border to move mesiad and in the majority of instances the right heart border to move laterad, though to a lesser degree. The movement of the right border may change again to an inward one which is probably induced by the tension of the pericardium. The diaphragm may descend far enough so that an area of decreased density is noted along the caudal heart border. This does not indicate a complete separation of the heart from the centrum tendineum though the area of contact is diminished. The pericardium along and immediately above its caudal insertion takes up the resulting space, with lung tissue following along its outer surface. The whole left contour becomes less curved. The vascular pedicle is elongated and narrower. The lateral view reveals, particularly in the costal type of respiration, an elevation and ventral movement of the sternal and cardio-vascular shadow; the retrosternal and retrocardiac spaces increase in size and transparency and the shadow of the inferior vena cava is better visualized.

Forced expiration shows a rise of the cardiac silhouette together with some displacement to the left. The angle between the main axis of the vessels and the heart decreases, and the waist of the left contour is more pronounced. A considerable portion of the cardiac silhouette fuses with the abdominal shadow. The vascular pedicle is shortened and becomes wider. The lateral view reveals the dorsal cardiac contour more curved; the shadow of the inferior vena cava is no longer visualized and the retrosternal and retrocardiac spaces diminish in size and transparency.

The cardiac shadow reveals a pendulum movement, alternating with inspiration and expiration. Whether this is an expression of an increased inspiratory filling of the right and emptying of the left heart or whether it is due to rotation is not definitely known, but it is very probable that the volume of the heart varies together with marked respiratory changes.

The degree of displacement is more marked during inspiration in the recumbent position and during expiration in the upright position.

Comparison of pairs of films taken during the inspiratory and expiratory phase of normal respiration shows that in the anterior view, all diameters as well as the cardiac area are more often increased than decreased during inspiration.

**INTRATHORACIC PRESSURE.** Marked changes, even though physiological, considerably influence the filling stage of the heart. The Muller test is performed by closing the glottis at the end of a full expiration and attempting an inspiration. It is followed by a rapid increase of the cardiac and superior vena cava silhouette. The Valsalva test is performed by closing the glottis at the end of a full inspiration and by forcing expiration. It leads to an increase in the intrathoracic pressure which in turn causes a rather slow decrease in the size of the cardiovascular silhouette, affecting all regions simultaneously. The degree of diminution is influenced by the amount of the muscular effort and probably depends also upon the condition of the heart muscle. The change in cardiac size from the Muller to the Valsalva test was computed in one instance to be 390 cm<sup>3</sup>. The peripheral arterial resistance also plays a rôle because there is noted a marked diminution of the heart size in hypoplastic types together with a marked fall in the blood pressure; while in physically well-trained persons the diminution may be less pronounced and the blood pressure may even rise to a considerable degree. The majority of individuals show a tachycardial reaction during the experiment while a few reveal slow, sluggish pulsations. Never do pulsations cease completely. As soon as the pressure is decreased, the heart size returns with a few large amplitudes to its normal size. The observation of the change of heart size is of interest because similar conditions are present in a number of physio-pathological conditions such as certain athletic performances, defecation, labor, cough and respiration of emphysematics and asthmatics.

**Lung Pattern and Hilus Region.** They are chiefly, though not exclusively produced by the blood-filled vessels, which fact was perceived by several men

monary vessels of one lung in a dog are tied off, the animal is sacrificed, the

lungs with the trachea are removed and the second lung is carefully rinsed. Then the lungs are inflated. The two sides vary in appearance. The ligated side shows a dense hilus shadow and extending from it solid ramifying shadows, with interspersed clear-cut, round, dense areas. The other side shows



FIG. 28—Arteriography (in the living). Appearance of normal arteries in popliteal region. Courtesy Prof. Dr. M. Sgaltzer, I. Chir. Univ. Klin. (Prof. Dr. E. Ranzi), Vienna.

very delicate shadows of little density. 2) the lungs with the trachea are removed from a body. With moderate inflation transparent bronchial lumina are noticed but no bronchial walls, with the exception of the immediate hilus region. With maximal inflation, the peripheral transparent ramifications disappear. The dense and ramified stripes appear; they represent blood

inflated lung and the inner surface thereby outlined in the proximal extent, no shadow can be demonstrated beyond the shadow of the barium sulphate which corresponds to a continuation of the bronchial walls 4) the transparent bronchial lumina may be seen within the collapsed lung of the living or within areas of infiltration. They disappear if normal conditions are restored 5) if a lung is collapsed by means of a pneumothorax, the contralateral lung soon shows a marked increase in its lung pattern 6) if roentgenograms are taken with the trunk dependent, the cranial portion of the hilus as well as all cranial solid shadows are markedly increased in size and density while the lung pattern toward the base is diminished. 7) the lung pattern increases markedly in the presence of certain diseases of the pulmonary artery system 8) the lung pattern may be decreased in the presence of stenosis of the pulmonary artery. 9) the lung pattern is increased in the presence of left-sided cardiac failure 10) if artificial changes in the intra-alveolar pressure are brought about, the lung pattern changes, it decreases with a decreased pressure and increases with an increased pressure 11) a roentgenogram taken at the beginning of the apneic pause demonstrates more pronounced markings than one taken after 20 seconds of apnea. 12) the size and density of the lung pattern is more marked when the film is made during ventricular systole and less marked when taken during diastole 13) in the presence of a valvular regurgitation of the pulmonary orifice, the larger solid shadows show definite expansile pulsations 14) if a contrast medium is injected by means of a catheter into the right heart, the pulmonary artery with all its branches is increased greatly in density. This has been depicted in human beings as well as in animals.

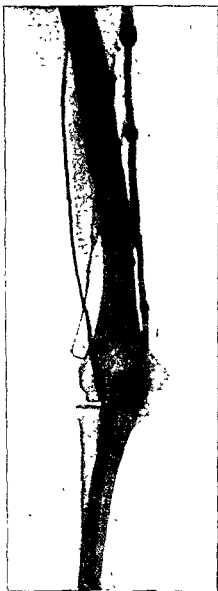


FIG 29—Venography (in the living) Appearance of normal veins and their valves Upper extremity, dependent Courtesy Prof. Dr M Sgaltzer, I Chir. Univ. Klin (Prof. Dr E Ranzi), Vienna



Transparent bronchial lumina are clearly seen in each hilus region. Farther away they are noted only in cross section, as a lumen surrounded by a small ring representing its wall. Due to superimposition and crossing, they blot out vascular shadows in part or completely. With the walls much thickened or with the lumina filled with secretion, visibility is increased. The lung vessels appear partly at the periphery and decreasing in size with a curved course and with dichotomic branching as spots of homogeneous density, along the course of solid shadows which fail to be reproduced in films taken in different positions of the patient. These spots represent vessels in cross section.

In short, it may be said that an increased air content of the lungs favors visibility of the blood vessels, a decreased air content favors visibility of the bronchial lumina.

The blood content in the lungs is subject to static displacement and it occurs most frequently in persons with postural circulatory deficiency on the constitutional basis of asthenia. It may be demonstrated roentgenographically since the apical vascular shadows are small for the erect position while they become larger, i.e., better filled with blood, in recumbency. The constitutional factor is modified by conditional factors such as menstruation, pregnancy, digestion, fluid intake or loss, weight loss, lack of sleep, debilitating diseases, and any factors that foster fatigue. It has been pointed out that a poor circulation through the upper portions of the lungs in the upright position would make these areas susceptible to infection with tuberculosis. It is interesting in this connection to remember that in adolescents and adults the cranial portions of the lungs are a favored site for the tuberculous infection while in infants and children location in the middle and basal portions is favored.

Each hilus has for its main elements arteries, veins, bronchi—all surrounded in the more distal portions by lung parenchyma. The visibility of both hilus regions increases in the presence of a rather narrow and vertical cardiovascular silhouette. The *left* main branch of the pulmonary artery becomes visible at the left at a level which corresponds to the most cranial point of the pul-

always be increased by turning the patient 10 to 20° toward the left anterior oblique view. It is seen, at a greater angle of rotation, within the window of the aorta. In the right anterior oblique view its origin is noted as a dense round or oval area, ventrad to the left main bronchus. The latter, in the anterior view, gives off its branch for the anterior (upper) lobe still within the cardiovascular shadow. In its further course, and in the presence of a small vertical heart, it is found to accompany the left heart border as a transparent band. The veins with cranial direction cross the descending main arterial branch. They are not uniformly visible. The *right* main branch of the pulmonary artery usually divides before the right border of the large vessel shadow is attained and the intersection of its cranial contour with the mediastinal border is more caudal as compared with the corresponding findings on the left. The shape

of the right hilus reminds one of the arrangement of the gray substance in one-half of a cross section of the spinal cord. The club shaped anterior (upper) horn corresponds to the division of the cranial lobe branch of the pulmonary



FIG 30—Aortography (in the living) Injection at level of first lumbar segment. Lower thoracic aorta, its branches and further ramifications are visualized. Courtesy Prof. Dr. H. Holfelder, Frankfurt a/M

artery; the frayed posterior (lower) horn corresponds to the common trunk for the middle and posterior (lower) lobe. The main branch is of a comma-like appearance and curves caudad. The right main bronchus subdivides into the anterior (upper) lobe and middle lobe branch with the right pulmonary

artery branch situated between. It continues its caudal course between the right border of the cardiac silhouette and the descending comma-like arterial branch, and, superimposed upon it in a slightly oblique course, blots out some of this vascular shadow. In the presence of a degree of right-sided enlargement, the bronchus can be seen through the heart shadow as a small transparent band. This is especially well noted in children. Some horizontal or oblique small vascular shadows may be present which cross arteries and bronchi horizontally or obliquely. They represent the right pulmonic veins.

The vena azygos may be visible more frequently in children than in adults in its horizontal course just before entering the sup vena cava. Its shape is egg-like, with a pointed upper and lower pole, and it is situated to the right of the trachea just above the right bronchus. Its transverse diameter measures 0.6 cm. in children, and 0.8-1.0 cm. in adults. The density of its shadow increases during inspiration and diminishes during forced expiration.

#### PERIPHERAL

Normal blood vessels, i e., the column of blood, can be demonstrated in the cubital and occasionally in the popliteal region, provided the exposures are taken with a very low voltage. The presence of thin muscular and larger fatty layers, occurring in women, favors this demonstration. When a radiopaque substance is injected into a blood vessel, it will be swept along with the current of blood, more swiftly and in a centrifugal direction in an artery; slower and centripetally in a vein.

**ARTERIES.** Normal arteries have a direct and largely longitudinal course and manifest a minimum of anastomoses with each other. The branches show an orderly arrangement and fine branches, usually few in number, are found to arise directly from the large trunks.

**VEINS.** The larger veins of the upper extremity show saccular or mushroom-like dilatations, corresponding to the level of the valves. This is best observed with the limb in the dependent position; the appearance of the valves changes, depending upon the direction of projection. The leaflets of these valves may occasionally be seen as linear defects within the contrast medium. Venous channels are observed connecting larger venous trunks. The direct injection usually demonstrates one or several larger superficial veins. Injection of superficial veins at the level of the ankle or knee, however, will fill some deeper channels by means of anastomotic branches. In order to demonstrate the deep venous trunks, the arterial injection with backflow into the veins is the method of choice. (As to observations of the blood flow, see chapter V.)

**LYMPHATICS.** Following a Thorotrast injection, the lymphatic vessels appear as very fine strands and the regional lymph nodes become visible. The former disappear within a few days while the latter remain visible because the contrast medium is stored. Such studies have been made not only on animals but also on man.

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## CHAPTER IV

### MEASUREMENT

Measurement (cm. and kg. will be used) is intended to establish standards for the normal and to decide whether or not a given case is within the range of normal variation and, if not, to give mathematically the degree of deviation. The term "range of variation" is preferable to the term "normal" and comprises all those findings which are most likely to be found in healthy people. From a mathematical viewpoint a heart larger than normal indicates merely that there is less probability of finding this size correlated with normal function. Our methods of measurement stress the formal-quantitative side of the problem and are insufficient as to the functional-qualitative aspect. It is therefore understood that shape and size found to be within the range of normal variation may well exist together with disturbed function. On the other hand, it must be admitted that a normal function is closely connected with a certain optimal size, at least if we think of function performed over long periods of time. Because of the latitude of the normal variation, no sharp distinction may be drawn between findings in the advanced normal range and those considered in the incipient abnormal stage. Borderline cases are therefore difficult to evaluate. It is to be remembered that a heart which is originally small may enlarge considerably before it exceeds the upper limit, while a heart which is near this limit to begin with will surpass it with very little increase. Further, the muscular wall may increase in thickness without producing appreciable alterations in shape and size of the silhouette.

The application of average figures is of limited value since we are dealing clinically with one individual and not with an abstract concept derived from a large number of individuals. Even when considering homogeneous groups, with little differences in weight, height, chest circumference, etc., and employing some of the best criteria, it will be found that, while the mean figures may be plotted in straight lines with even progression of values for each increment in the criterion, the individual figures are dispersed through a considerable range. If several such homogeneous groups are studied, arranged in ascending or descending progression, it is usually found that the maximum and minimum figures in each group fall beyond the average figures of one or more of the adjacent groups. To give an example, a group of individuals has been subdivided into weight groups of 5 kg. and chest circumference groups of 5 cm. One group represents persons with a weight of 55-60 kg. and a chest circumference of 80-85 cm.; for a second group the respective figures are 75-80 kg. and 95-110 cm. The average figures for a diameter or for the area of the silhouette will be smaller in group 1 as compared with group 2; but the maximal figures in group 1 may be superior to the minimal figures in group 2. The use of absolute figures likewise is of little practical use, especially when considering the influence of age and sex. Such absolute figures must be correlated with other characteristics of an individual. The mathematical treatment consists of obtaining partial and multiple coefficients of correlation and

of giving to each variable a weight that will produce the highest degree of correlation between the variables and the criterion. The final purpose is to produce a prediction formula and to make a statement of this kind: If the deviation from the predicted figure is . . . the degree of probability that we are dealing with an abnormal finding is . . . A finding of +10% in a healthy, physically well-trained person has less significance than the same finding in a sedentary person with a slight increase in blood pressure. Certain difficulties have arisen in correlation measurements; if one correlates the area of the heart silhouette with the weight of the body ( $\approx \text{volume} \times \text{specific weight}$ ) one may not find the expected correlation, while if one uses the volume of the heart, one will.

The type of material from which the standards for prediction tables and formulas were derived should be known: the standards derived from people in first class physical condition and training are not quite the same as those of the average population; racial characteristics of build also should be considered. The applied roentgenologic method should likewise be known. One cannot compare orthodiagraphic with teleroentgenographic standards. Suppose that one has available an orthodiagram and a teleroentgenogram taken instantaneously at 2 m. distance for each member of a group of healthy individuals. As usual, the former examination is carried out at the endphase of tidal respiration and the latter at a moderate degree of inspiration. It is furthermore assumed that the measurements obtained on the teleroentgenogram are corrected on the basis that the object-film distance is 75 cm. In determining the transverse diameter (T) by the two methods, a coincidence within  $\pm 0.5$  cm is noted for about half of all the cases. But T in the teleroentgenogram is found to exceed T in the orthodiagram by as much as 1.8 cm. and, on the other hand, T in the orthodiagram is noted to exceed T in the teleroentgenogram by as much as 2.4 cm. Such extreme discrepancies probably would be less were teleroentgenograms taken at the same predetermined point of the cardiac cycle and if an involuntary Valsalva effect could be safely excluded. Suffice it here to say that the computation of orthodiagraphic size from corrected teleroentgenographic size, or vice versa, is not permissible. One may not compare results obtained, even though by the same method, if there was a different phase of respiration or a difference in body position. The necessity of exactness when gaining and comparing figures is perhaps clearly illustrated by the fact that a sphere of 10.3 cm. in diameter has a volume of 10% more than one with a diameter of 10 cm. Or to give another example. A heart that has oblique, broad and depth diameters of 13, 10 and 9 cm, respectively, will have a computed volume of 526.5 cm<sup>3</sup>. Add 0.5 cm. to every measurement and the volume becomes 609 cm<sup>3</sup>, which is a 15.7% increase. Such facts are well to bear in mind when one attempts to compare heart volumes by means of roentgenograms for a given person. While double determinations in the same cardiac phase accord well, the difference between diastolic and systolic volumes will vary from 9-23% with an average of 15%.

When examined at different times the normal heart and vessels are noted to be constant in shape and size, provided the following criteria are identical: phase of cardiac cycle and respiration, body position and heart rate. When changes occur, they are found to be of minor degree and of short duration.

and should be attributed to changes in filling. The change from standing to sitting position increases the volume of the heart by an average of 10%, and the change from the standing to the recumbent position will frequently produce a volumetric increase of 250 cm<sup>3</sup>.

Only two methods of roentgenologic measurement are anatomically exact: one determines the diameter of the arch of the aorta, the other is represented by the volumetric reconstruction of the heart. Other methods are inexact, such as the measurement of the ascending aorta; or the determination of the cardiac area which requires an artificial construction. The diameters which are commonly used do not correspond to any actual anatomical diameters, they

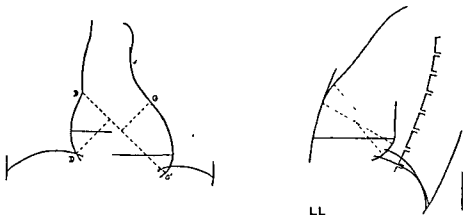


FIG 31—Nomenclature in the anterior and lateral views D—intersection of vascular and cardiac contours at rt D'—intersection of cardiac and diaphragmatic contours at rt G—atrio-ventricular border at lt, to be determined by fluoroscopy G'—farthest point at lt, lower pole region Long or oblique diameter (L) connects points D and G' (dash line). Broad diameter (B), sum of lengths of 2 perpendiculars dropped from points G and D' respectively to L (dash-dot line) Transverse diameter (T), sum of maximal horizontal distances of rt and lt silhouette borders to mid-sternal line (solid line) G-G'—lt. ventricular chord, G'-D'—rt ventricular chord (dotted lines)  $T_{D-hor}$  connects horizontally the most distant dorsal point of the silhouette with the ventral inner chest contour (solid line).  $T_D$  connects obliquely the farthest dorsal point of the silhouette with the ventral inner chest contour (solid line).

are located in planes with different angulation and are practically never vertical to the direction of projection. We are dealing with the two-dimensional projection of a three-dimensional, irregular body, the shape and position of which vary from case to case.

The recent introduction of contrast visualization has made possible to a certain degree, the measurement of the height, width and thickness of the chamber walls and the length and thickness of the vascular walls.

The available anatomical data are of but little use for the roentgenologic approach. F  
the anatom

refer to the capacity of the chambers and not to the sum of the capacity plus muscle mass.

Measurements and figures will refer, unless otherwise noted, to the orthodiagram and standing position (see figure 26).

A rather lengthy presentation of the available data seems necessary; partly because it is nowhere else available, partly in order to justify the conclusions at the end of this chapter.

**Measurement of Cardiac Shadow. ABSOLUTE FIGURES, LINEAR, AREAL, VOLUMETRIC. Anterior View.** 1) The long or oblique diameter (L) is the

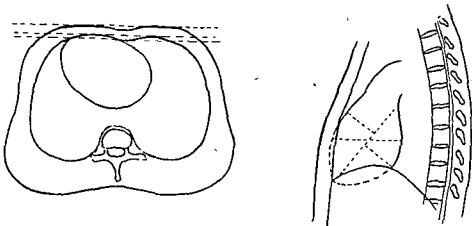


FIG. 32—Schematic horizontal and sagittal section through the chest to demonstrate the advantage of using the oblique rather than the horizontal depth diameter of the heart. The sternum is physiologically slightly depressed in the majority of cases. Hence a line that is drawn tangentially at the ventral contour of the heart may lie slightly in front of a line that is drawn tangentially at the dorsal contour of the sternum. The horizontal depth diameter is obtained by drawing in the lateral view a line that connects the dorsal contours of the cardiac and sternal shadows. It is thus assumed that the ventral cardiac and dorsal sternal contours coincide. Since this is frequently not the case, the depth diameter of the heart may be actually greater. The oblique diameter, which is the sum of the two lines drawn perpendicularly on a line that approximately connects the bifurcation of the trachea with the ventral costo-diaphragmatic junction, has its ventral starting point at a clearly defined border of the heart. Adapted from J. Palmieri, G. G., *Bull. Sci. Med.*, 1939, 111, 482.

distance from the junction of the cardiac with the vascular silhouette at the right (point D) to the farthest point at the left lower pole (point G'). The exact outline of this region must be visible. While difficulty is often encountered in determining accurately, in the roentgenogram, the contour of the left lower pole, it is almost always possible to trace this contour orthodiagraphically during fluoroscopy. The average for the adult male is 13.6 cm. The approximate minimal and maximal figures are respectively 11 and 15.5 cm.—Anatomical measurements on hearts of 275 subjects who had died quite suddenly and without cardiovascular disease, showed an average figure of 12.6 cm. for L.

The broad diameter (B) represents the sum of the lengths of two perpendiculars dropped to the line of the long diameter; one from the junction

of the right lower border with the diaphragm (point D') and the other from the junction of the left auricular, pulmonic artery or pulmonary conal arch with the left ventricular border (point G). The average for the adult male is 9.8 cm. The approximate minimal and maximal figures are respectively 8 and 11.2 cm.

The transverse diameter (T) represents the sum of the maximal distance of the right and left silhouette borders to the midsternal line. The average for the adult male is 12.2 cm. The approximate minimal and maximal figures are respectively 9.2 and 14.5 cm.

All these figures are on the average 1.2 cm. less for the adult female; but when related to the same weight the difference is found to be less, namely, 0.2-0.5 cm.

The respective average figures for the newborn (teleroentgenogram, recumbent position) are 5.4 cm. for L, 4.0 cm. for B and 5.1 cm. for T.

The left ventricular chord (LV), from G to G' subtends the arc of the left lower ventricular silhouette. The average, minimal and maximal figures for the adult male were found to be 9.6 (6.6-12.5) cm., for the adult female 8.5 (6.0-10.5) cm. The right ventricular chord (RV) connects G' and D'.

Normally the following size relations exist  $L > T > B = RV > LV$ .

The angle of inclination is formed by the limbs L and a horizontal line from G' toward the midline (some choose to determine this angle as formed by L and the midline). The average angle is about  $43^\circ$ , hearts with a definitely greater angle, and as much as  $57^\circ$  was found, are vertical hearts, those with a definitely smaller angle, and as little as  $20^\circ$  was found, are horizontal or transverse hearts. T is usually in a reverse ratio to this angle. Formulas in which T is used increase in reliability when the value of the angle is also introduced.

2) The area ( $A\text{-cm}^2$ ) is obtained by the planimetric determination of the completed silhouette: the cranial and caudal limits of the cardiac silhouette are drawn by rather conjectural delineation. The caudal border is often drawn too flatly and with the conventional cranial border a part of the conus arteriosus is lost. While area determinations by the same individual, but at different times, show an error of 2-5%, it is usually greater when done by different examiners and differences as high as 20% may be found. The average and approximate minimal and maximal figures for the adult male (orthodiagram, standing position) are 102 (75-130)  $\text{cm}^2$ , for the adult female 90 (60-110)  $\text{cm}^2$ . The figures for the area as projected into the horizontal plane are almost the same. In the recumbent position the figures are on the average 20% greater. The area in the newborn varies from 15-17  $\text{cm}^2$ .

The heart silhouette has been considered as an ellipse; the area then is the product of the perpendicular long and short diameters times 0.7854 ( $\pi/4$ ); as compared with the area the variation was found to be 2.6-6.9%. The high correlation between the area and the  $L \times B$  product (see later) has led several authors to use  $L \times B$  instead of the area.

3) The index of depth determines the depth of a point on the dorsal surface of the left ventricle. With a target screen distance of 60 cm. the extreme point on the left lower contour is marked orthodiagraphically. The tube is then shifted 10 cm. toward the midline and the projection of an extreme left point is marked again. The distance corresponds to the index; its upper limit is 1.4

cm. An increase would indicate an increased dorsal prominence of the left ventricle. However, the index is influenced by several other factors; it changes with horizontal and vertical displacement. And misleading figures are also obtained when the distance is too great from the left lower pole portion to the screen. In order to correct this, one must either increase the standard target-screen distance or diminish the standard lateral shift, or both.

4) A depth diameter was determined in the silhouette projected into the horizontal plane, its figures varied for adult males from 9-11.5 cm. and for adult females from 7.3-10.0 cm.

*Oblique and Lateral Views.* 1) In order to examine the heart in the direction of its main axis, an angle of  $20-35^\circ$  in the right anterior oblique view is chosen. To see the heart perpendicular to its other axis, an angle of  $55-70^\circ$  in the left anterior oblique view is used. A vertical line has been drawn within the silhouette in the left anterior oblique view in an attempt to represent the interventricular septum. But neither is there agreement as to the most desirable angle nor are the landmarks for the cephalic end of this line certain; in addition, the septum lies in a curved plane.

2) Different depth diameters have been determined in the lateral view; symbols for them are not as yet standardized and those used here are suggested for acceptance.  $T_{D-hor}$  is drawn from the most distant dorsal point of the silhouette horizontally to the frontal inner chest contour. It should be noted that this contour is not quite identical with the anterior cardiac contour. If frontal planes are laid through these contours, the latter are found to be somewhat more ventrad than the former. This fact is clearly demonstrated by volumetric reconstruction. The average, minimal and maximal figures for the adult male (orthodiagram, standing position), as given by three different authors are 9.4 (7.6-10.9), 9.6 (7.1-11.6) and 9.0 (8.1-9.9) cm. For the adult female one investigator found 8.3 (7.0-10.5) cm.  $T_D$  encloses an angle with the horizontal line; it connects the most distant dorsal point with a point just below the junction of the ventral cardiac contour with the sternal shadow. It varies for adult males from 6.7-10.7 cm., with an average of 8.7 cm., for adult females from 6.3-9.6 cm., with an average of 7.8 cm.  $T_{D-br}$  is constructed similarly to B in the anterior view. It represents the sum of the lengths of two perpendiculars dropped from the farthest ventral and dorsal contours respectively to a line which connects the caudal end of the trachea with the sternodiaphragmatic angle. The average, minimal and maximal figures for adult males were found to be 9.3 (7.1-11.5) cm., the average, minimal and maximal figures for the angle enclosed between this and a horizontal line, are  $45^\circ$  ( $25-67^\circ$ ).

3) The angle of disappearance determines the degree of dorsal development of the left ventricle. The patient is rotated from the posterior into the right posterior oblique view, with the right shoulder as a pivot, the left shoulder moving increasingly away from the screen. The shadows of the heart and spine travel in opposite directions until at a certain angle the extreme left heart contour just disappears in the spinal shadow. This angle may be measured by placing and fixing a rod on the patient's back just below the scapular angles. Two small lead crosses are attached at a distance of 20 cm. When the desired angle of obliquity is reached, the distance of the lead marks is determined orthodiagraphically and if divided by 20, the cosine of the angle is

obtained. The upper limit of this index is 35°. There is but little value in this procedure because extrinsic factors interfere, such as the curvature of the spine, the respiratory phase, air in the stomach, displacement of the heart to the left, etc.

*Volumetric Determinations.* These may be actual as a volumetric reconstruction described in Chapter I. The volume is obtained by immersion of the reconstructed model in water and is found to be 600-700 cm<sup>3</sup>. for the male and 450-500 cm<sup>3</sup>. for the female. On such a model the individual shape of the heart may be clearly seen, and the direction of the axis of the heart, connecting the region of the entrance of the inferior vena cava with the apical pole region, can be determined. It encloses, in the horizontal plane, an angle with the antero-posterior axis, which is open ventrally and shows an average of 42°. The other, much less exact method, involves the use of formulae, if such formulae are used they should be tested in comparing the results with the actual figures obtained by reconstruction in the living. This has been done and the following formulae permit an *approximate* determination of the cardiac volume. 1)  $V = 1.33 \times A \times T_{D-hor}$  (taken in the lt ant obl view) The error was found

to be from +7.6% to +24.0%. 2)  $V = \frac{a+b}{2} (bc + ca + ad + db)$ ;

( $a + b = B$ ;  $c$  and  $d$  equals the distance from  $D$  and  $G'$  respectively to a point on  $L$  midway between the base points of both  $B$  lines); the heart is considered as made up of 4 regular ellipsoids with a depth equaling  $a + b$ . This formula simplified reads  $0.5236 \times L \times B^2$ . The maximal deviations were found by one investigator to be -7.3% and +26.6%, by another, -12.2% and +20.9%. The weakness of this formula is that it assumes the depth of the heart—the antero-posterior dimension—to be always in an identical ratio to the diameter  $B$ . This is not so and a figure of +20.9% was actually found for a heart which was relatively large and flat 3)  $V = 0.45 \times L \times B \times T_{D-hor}$ . The average, minimal and maximal figures for this value for males, 19-25 yrs, were 569 (407-835) cm<sup>3</sup> 4) Another formula was tested by actually determining the volume in well filled and hardened postmortem specimens and roentgenologically,  $A$  and  $T_{D-hor}$ . It is based upon the following principle. The volume of any regular body may be determined if the size of its parallel projection and its greatest diameter in the direction of projection are known; it equals the product of these factors multiplied by a constant, which depends upon the geometric form of the body. It is 0.667 for an ellipsoid, 0.59 for a paraboloid. Choosing a figure in between the formula reads  $V = 0.63 \times A \times T_{D-hor}$ . This formula is considered to have a maximal error of 15%. The average figures for a group of adult males were 630 cm<sup>3</sup>, for a group of adult females 487

were 387

for female

grams wei

the enlargement of the heart shadow on the film with the constant factor being 0.67, the minimal and maximal figures were noted to be 457 and 963 cm<sup>3</sup>. Related to the same body weight (50-60 kg.), a male group was found to exceed a female group by an average of 75 cm<sup>3</sup>. Another investigator noted the heart volume of females to be 15-25% smaller as compared with males of the



same weight and width of chest. 5)  $V = 0.53 \times A^{3/4}$ . 6)  $V = 0.64 \times A^{1.45}$ . These two formulae were derived from combined roentgenological and anatomical studies on cadavers.

The comparative value of the two equations referred to in the preceding paragraph under 3) and 4) has been tested as follows. Plastic heart models were made from 20 healthy individuals and from 10 bearers of heart disease. These models were a) submerged in water in order to determine the actual volume, and b) teleroentgenographed for the anterior and lateral views after the end points of the diameters had been identified on these models by means of lead marks. The extreme deviations from the actual volume figures were as follows:

Equation for V	Normal	Hearts Diseased
$0.45 \times L \times B \times T_{D-br}$	+8%, -8%,	+15%, -10%,
$0.63 \times A \times T_{D-hor}$	+16%, -1%,	+42%, +4%.

Therefore the formula  $0.45 \times L \times B \times T_{D-br}$  is recommended.

**CORRELATIONS BETWEEN ABSOLUTE FIGURES OF CARDIAC SIZE AND BETWEEN THESE FIGURES AND CERTAIN BODY MEASUREMENTS.** The coefficient of correlation ( $r$ ) will be mentioned repeatedly. A correlation of zero denotes a complete lack of relationship and signifies that one measure is of no value in predicting the other. A correlation coefficient of one or minus one denotes complete mutual interdependence, directly and inversely respectively. Figures for the standard deviations have been omitted for the sake of brevity.

Unless otherwise noted, figures refer to the examination in the upright position and to orthodiagrams.

The results which have been obtained by a number of investigators are given first for the static-anatomical aspect, to be followed by a functional-physiological approach.

*Anterior view. I. Correlation between figures of cardiac silhouette, cardiac volume, cardiac angulation.*

1) Diameters and diameters. B is 65-88% of L with an average of 75%. This is in good relation to the anatomical figure of 80% as obtained in a great number of normal hearts. B prevails in silhouettes commonly encountered with the constitutional form of the longtype. L prevails in the brachitype.—

The average figure for  $\frac{G'D'}{G'D}$  is for 5 years of age 1.43, for 10 years 1.33,

for adults 1.22.

2) Diameters and area. For the recumbent position, the areas of different silhouettes were found to be related as the squares of the respective L figures, with an error of not more than 10%.—There is a high correlation between  $L \times B$  and A ( $r = 0.85$  and  $0.94$  by two authors) and the  $L \times B$  product (heart rectangle) may be therefore used instead of A. But it should be realized that  $L \times B$  on the average equals  $A \times 1.38$ . The shape of the silhouette, as expressed by the  $\frac{L}{B}$  ratio has no correlation with A, i.e., one may find smaller as well as larger areas in different constitutional types.

3) Diameters, areas and inclination. The coefficient between the angle of inclination with L is zero, with B — 0.21, with A 0.029 and with T 0.53; i.e., T depends not only upon the size but in a far-reaching degree upon the position of the silhouette. Formulae and tables which use T exclusively are therefore of limited value.

II Correlation of figures of cardiac silhouette with certain body measurements

a) With Weight (W) and Height (H).

W and H appear in most prediction formulae. The importance of H has been overestimated, mainly because it possesses only one-third of the variability of W. H is a valuable criterion only when it is found together with a proportional general body development. There is an advantage in retaining H in formulae because the errors introduced when it is omitted accumulate in obese subjects. Height groups were found to be better criteria than age groups for children. W is a better criterion than H but fails in the presence of obesity; also the heart is found to be relatively smaller in people with a great body mass (megalosomics).

1) Diameters T correlates better with W than with H. For 2300 selected individuals, age 20-30 years,  $r$  for W was 0.40-0.57 (four authors), for H 0.12-0.21 (three authors). For people taller than the average for weight, T tends to indicate smaller hearts while for people shorter than normal for weight, it tends to indicate larger hearts. This is explained by the value of T depending much on body build. T in children (teleroentgenograms, distance 182.5 cm) expressed in % of H was found to decrease on the average from 8.4% at 2 years to 6.8% at 13 years. T correlated with W, in infants, not better than 0.244.—The correlation coefficients for W and H respectively were by another author found to be for L 0.385 and 0.287, for B 0.311 and 0.434, B correlates somewhat better with H, L somewhat better with W. For  $T_{D_{hor}}$  and W,  $r$  is 0.36 and for  $T_{D_{hor}}$  and H,  $r$  is 0.22.—A prediction formula for T is:  $0.1094 \times \text{Age} - 0.1941 \times H + 0.8179 \times W + 95.8625$  with  $r$  0.588 and 0.693 respectively in two studies. Prediction formulae for T in children and adolescents (teleroentgenograms, distance 182.5 cm with correction for distortion due to divergence) are  $0.0637 \times W + 7.5$  and  $0.00490 \times H + 0.0569 \times W + 7.1$ , with  $r$  0.81 in both instances. In the following average ratios, figures in the brackets indicate the ratios obtained in the recumbent

position  $\frac{B \times L}{W} = 2.01 (2.51)$ ,  $\frac{B \times L}{H} = 0.746 (0.934)$ .

2) Area and diameters  $r$  for W and A is 0.480,  $r$  for H and A is 0.313; A, like L, correlates better with W.—A prediction formula for A in adults is:  $0.0204 \times \text{Age} + 0.8668 \times H + 0.337 \times W - 63.8049$  with  $r$  0.578 and 0.547 respectively in two studies. For children and adolescents beyond the age of 2½ years and below the height of 160 cm. A equals  $0.18 \times H + 1.045 \times W + 13.7$  (teleroentgenograms, distance 182.5 cm. with correction for distortion due to divergence),  $r$  was found to be 0.925. This formula has been tested by another author who found that in 47% of a group of normal children the actual area was 11-30% greater than the predicted area.—Here it may be remembered that until puberty there is little difference in the size

of the heart between girls and boys.—It has been suggested not to correlate A directly with W but with its  $\frac{2}{3}$  power. It was found for children at the age of 6-12 years that  $\frac{A \times 100}{W^{2/3}}$  equals 722-788 with an average of 750; in stout

and edematous children this quotient tends to be smaller, in undernourished children higher. In 5% of normal children, however, abnormal figures were found. The relation of A to W deserves further study.

The exponent for W in its relation to heart weight has been determined in a larger anatomical series and the average somatic exponent was found to be 0.8181 for males and 0.7247 for females, the anatomical material, however, was not without objection since W was abnormally low in a number of instances because of preceding diseases of long duration.

In the following average ratios, figures in the brackets indicate the ratios obtained in the recumbent position;  $\frac{A}{W} = 1.51$  (1.91);  $\frac{A}{H} = 0.563$  (0.71).

b) With horizontal chest diameters, computed volume of chest and trunk, size of fist.

Unless otherwise mentioned, Th is measured orthodiagraphically and diagonally between both inner chest contours; some draw it at the level of the costophrenic sinuses, a few at the level of the dome of the right diaphragm.

1) Diameters. The correlation coefficient of B and Th (external transverse chest diameter at the level of the 4th rib) is 0.32, of L and Th 0.408.—In the following average ratio the figure in the bracket indicates the ratio obtained

in the recumbent position:  $\frac{B \times L}{H \times Th} = 0.3$  (0.356) (Th taken ortho-

diagraphically at the 4th interspace). For children beyond the age of 2 years (teleroentgenograms, distance 150 cm.),  $\frac{H \times Th}{B \times L}$  equaled on the average 29.3;

81.5% of all cases showed a variation from 24.6-34.0 and 92.5% from 23.2-35.5.—The correlation coefficient for T and Th was found by one author to be 0.435 (Th taken as an external measurement at the level of the 4th rib), by another one 0.28 (Th taken orthodiagraphically at the level of the costodiaphragmatic sinuses). For the recumbent position the figures for the T:Th ratio varied from 42%-55%. Two different investigators found the range of variation (teleroentgenogram, distance 182.5 cm.) 36%-57% with an average of 45% and 49.5% respectively. For a large group of children (partly teleroentgenograms, distance 182.5 cm., and partly orthodiagrams) the range of variation for  $\frac{T}{Th}$  of the cases was 44-50%, for  $\frac{1}{3}$  41-54% and in 25% the ratio was above 50%. The "cardio-thoracic ratio" should therefore be con-

stant size.  
circumference) is 0.220, of  
ing average ratios, figures

in the brackets indicate the ratio obtained in the recumbent position:  
 $\frac{B \times L}{Cir} = 1.41$  (1.765);  $\frac{B \times L}{H \times Cir} = 0.831$  (1.038).—One prediction formula

for T reads:  $\frac{W}{120} + \frac{H}{90} + \frac{Cir}{30} + 0.3$ . Another prediction formula for T is:

$-0.0593 \times \text{Age} - 0.9143 \times \text{H} + 0.9594 \times \text{W} - 0.2095 \times \text{ant.-post. ext. diameter of chest} + 0.0256 \times \text{lat. ext. diameter of chest} + 0.4986 \times \text{girth} + 208.2$  (chest measurements taken at level of third intercostal space);  $r$  was found to be 0.681.—Similarly as to the T:Th ratio, it was found that the D'G':Cir ratio shows marked individual variations and is much influenced by body build; while small figures for D'G' (7-8 cm.) occurred only with Cir less than 80 cm., and high figures (14-15 cm.) with Cir only above 90 cm., there was no parallelism found for the range of 80-90 cm. for Cir.

It is necessary now to define the terms chest and trunk value, as used in

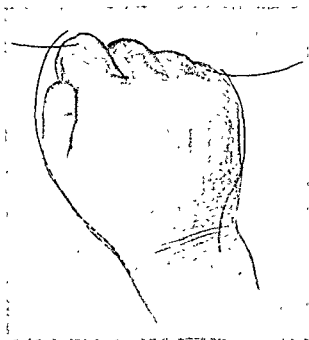


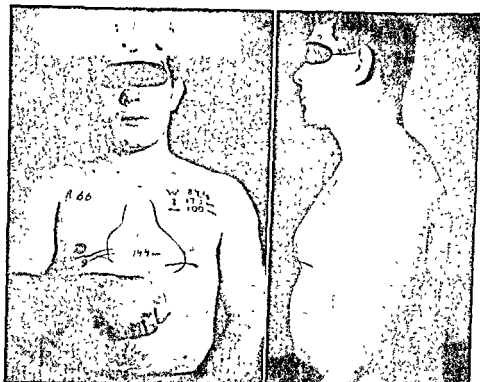
FIG. 33.—This drawing shows how to compare the size of a patient's cardiac silhouette with that of his clenched fist. The cardiac silhouette was drawn orthodiagraphically, copied on a semitransparent sheet, and the sheet was rotated by about  $180^\circ$  to enable the patient to conveniently place his fist in the silhouette.

An approximate agreement of the cardiac area with the size of the fist is noted. Hence cardiac enlargement is excluded.

anthropometrics. The chest value is expressed by the product of height of sternum  $\times$  transverse chest diameter  $\times$  antero-posterior chest diameter (both taken at the level of the 4th rib). The trunk value consists of the sum of the chest, upper and lower abdominal values. The upper abdominal value is expressed by the product of xipho-epigastric distance  $\times$  transverse hypochondrium diameter  $\times$  antero-posterior hypochondrium diameter; the lower abdominal value is expressed by the product of pubo-epigastric distance  $\times$  transverse pelvic (bicrestal) diameter  $\times$  antero-posterior hypochondrium di-

ameter For B and chest value  $r = 0.312$ , for trunk value  $r = 0.341$ ; for L and chest value  $r = 0.231$ , for trunk value  $r = 0.301$ ; for T and chest value  $r = 0.233$ , for trunk value  $r = 0.343$ .

Laennec studied the close anatomical relationship between the size of the heart and the fist. It was noted that the ratio of L to the length of the clenched



9 yrs of football, baseball and  
circumference 100 cm Apical  
B 10, Th 24.8 cm Transverse  
within normal limits, not ex-  
us instance

fist (distance from the metacarpo-phalangeal joint of the 5th finger to the inter-phalangeal joint of the thumb) equals 1.39-1.56

2) Area. The correlation coefficient of A and Th (external transverse chest diameter at the level of 4th rib) is 0.421.—A prediction formula for A is: —  $0.6132 \times \text{Age} + 0.0504 \times H + 0.5787 \times W - 0.1805 \times \text{ant-post. ext. diameter of chest} + 0.1601 \times \text{lat. ext. diameter of chest} - 0.2876 \times \text{girth} + 81.18$  (chest measurements taken at level of third intercostal space);  $r$  was found to be 0.575.—In the following average ratios the figures in the brackets

indicate the ratios obtained in the recumbent position:  $\frac{A}{H \times Th}$  (Th taken orthodiagraphically at the 4th interspace) = 0.226 (0.27),  $\frac{A}{Cir} = 1.059$

(1.34);  $\frac{A}{H \times \text{Cir}} = 0.625$  (0.789).—For children beyond the age of 2 years (teleroentgenograms, distance 150 cm.)  $\frac{H \times \text{Th}}{A}$  equaled on the average 39.3; 81% of all cases showed a variation from 33.3-45.3 and 91.5% from 31.4-47.2.—The coefficient of A and Cir was 0.332, of A and surface of chest area

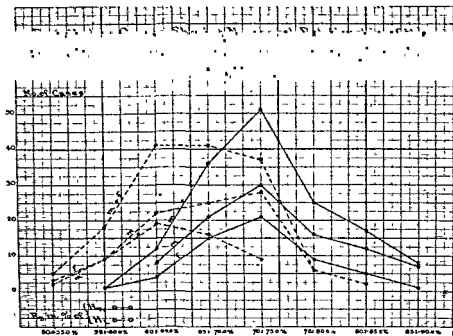


FIG. 35.—The graphs represent distribution curves. The number of cases are plotted on the abscissa; on the ordinate the depth diameter of the heart ( $H_D$ ) taken in the lateral view, expressed in per cent of the transverse diameter ( $H_T$ ) and of the oblique or long diameter ( $H_L$ ) both taken in the anterior view. The solid lines represent the  $H_D/H_T$  ratio, the broken lines the  $H_D/H_L$  ratio. m = male, f = female. From Roesler, H., *Am Journ Roentg*, 1934, 32, 464. Courtesy Charles C Thomas, publisher.

0.314. For A and the chest value, r was 0.258, for A and the trunk value, r was 0.373.

It is advised, for practical purposes, to place the right fist of the individual over the orthodiagraphic silhouette so that L is approximately identical with the line connecting the proximal and ulnar end of the fist with the interphalangeal joint of the index finger. For this purpose the sheet on which the silhouette was drawn is turned by approximately  $180^\circ$ . One finds that under normal conditions the outlines of fist and silhouette almost coincide.

c) With the surface area and age

The surface area of the body equals  $W^{0.425} \times H^{0.725} \times 71.84$  and most investigators agree that there is no practical advantage in using it. One author, however, found the r for body surface A and A as high as 0.84-0.92. In another

ameter For B and chest value  $r = 0.312$ , for trunk value  $r = 0.341$ ; for L and chest value  $r = 0.231$ , for trunk value  $r = 0.301$ ; for T and chest value  $r = 0.233$ , for trunk value  $r = 0.343$ .

Laennec studied the close anatomical relationship between the size of the heart and the fist. It was noted that the ratio of L to the length of the clenched

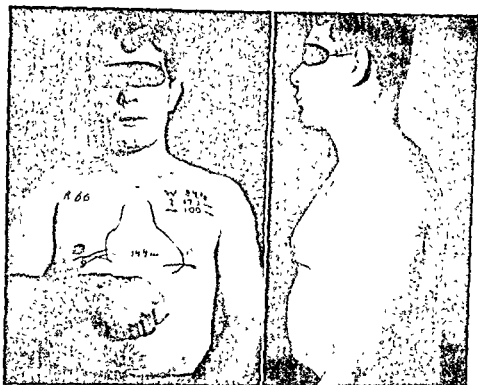


FIG 34—22 yrs, M No complaints. No history of infection 8 yrs of football, baseball and hockey Athletic type Height 170 cm., weight 84 kg., chest circumference 100 cm Apical thrust barely palpable Rate 66 B P 138/80 L 147, T 144, B 10, Th 24.8 cm Transverse type of heart with relatively high position of diaphragm Heart within normal limits, not exceeding size of fist. The T/Th ratio would be misleading in this instance

fist (distance from the metacarpo-phalangeal joint of the 5th finger to the inter-phalangeal joint of the thumb) equals  $1.39-1.56$ .

2) Area The correlation coefficient of A and Th (external transverse chest diameter at the level of 4th rib) is 0.421.—A prediction formula for A is:  $-0.6132 \times \text{Age} + 0.0504 \times H + 0.5787 \times W - 0.1805 \times \text{ant.-post ext diameter of chest} + 0.1601 \times \text{lat ext. diameter of chest} - 0.2876 \times \text{girth} + 81.18$  (chest measurements taken at level of third intercostal space);  $r$  was found to be 0.575.—In the following average ratios the figures in the brackets

indicate the ratios obtained in the recumbent position  $\frac{A}{H \times Th}$  (Th taken orthodiagraphically at the 4th interspace)  $= 0.226$  (0.27);  $\frac{A}{Cir} = 1.059$

study the quotient of  $\frac{\text{body surface A}}{A}$  showed a mean of 17.2 for normal children; 98% of all cases showed a lower limit of 14 — Age is not significant as a correlation factor for the adult. For children H and W are used preferably.

*Left Anterior Oblique View.* The range of variation for the ratio of the depth diameter ( $40^\circ$ - $45^\circ$ ) to T (ant view) was determined as 1.1-1.4 with an average of 1.25; the ratio of this depth diameter to a chest diameter, drawn from the projection of the anterior, right chest wall to the projection of the left costovertebral articulation, was found to be on the average 2.02 with a range of variation from 1.8-2.3 (teleroentgenograms, distance 182.5 cm.).—At an

	Case 1 Fig 36	Case 2 Fig 37			
Age	24 yrs	28 yrs.			
Height	167 cm	177 cm			
Weight	63 kg	68 kg			
Chest circ.	81 cm	89 cm			
L	12.4 cm.	12.5 cm	}	..	cardiac silhouette .. (1)
B	10.6 cm.	8.9 cm			
T	12.8 cm	10.1 cm.			
L + B + T	35.8 cm.	31.5 cm			
T <sub>D</sub>	7.3 cm	9.2 cm	}	..	" .. .. (2)
L × B × T <sub>D</sub>	959.5	1023.5			
$\frac{T_D}{L}$	55.8%	73.6%	min.	55.0%	} .. " .. (3)
			av	66.3%	
			max	75.0%	
$\frac{T_D}{B}$	68.8%	103.4%			" .. (4)
$\frac{T_D}{T}$	57.0%	91.1%	min.	62.5%	} .. " .. (5)
			av	73.0%	
			max	85.0%	
Th	26.2 cm	23.8 cm	}	av	chest silhouette .. (6)
Th <sub>D</sub>	6.4 cm	11.8 cm			
$\frac{Th_D}{Th}$	24.4%	49.5%			
$\frac{Th_{ext}}{Th}$	30.5 cm	26.0 cm	}	..	" .. (7)
$\frac{Th_{D-ext}}{Th}$	14.0 cm	24.5 cm			
$\frac{Th_{D-ext}}{Th}$	45.9%	94.3%			

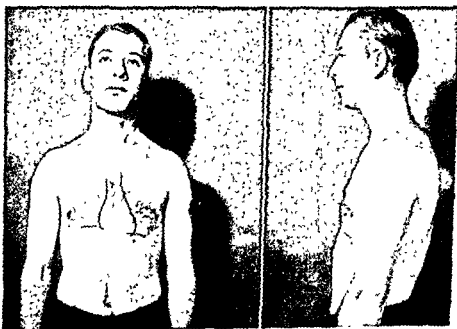
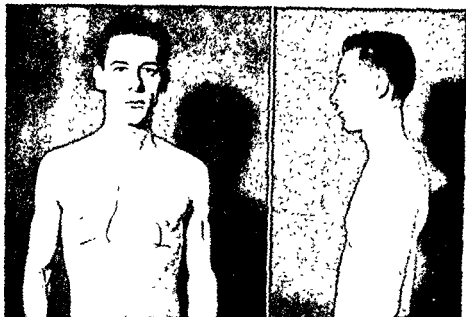
The cardiac silhouette of case 1 is larger than that of case 2 (ant view) . . . (1—The cardiac volume of case 1 is certainly not larger but most probably smaller than that of case 2 . . . (2 (The  $L \times B \times T_D$  product has a direct relation to the actual heart volume)

The heart of case 1 is rather large (ant. view) but shallow (lat view); that of case 2 rather small (ant. view) but deep (lat. view) . . . (3, (4, (5

The chest of case 1 is shallow, that of case 2 deep (lat views) . . . (6, (7.

These two cases represent types at either end of the normal distribution curve for heart and chest shapes.





FIGS. 36, 37.—Two healthy medical students, one with a shallow, the other with a deep chest. The intimate formal relationship between heart and chest types is shown in the following figure. (For interpretation of symbols in all other chest measurements.)  
 at level of 1  
 verse diamet  
 $Tb_{st}$ —sterno-  
 chest,  $Th_{st}$   
 series (see Fig  
 case 2 bottom

with L ( $r = 0.275$ ) and with B ( $r = 0.203$ ). Better, however, is the correlation of  $\frac{T_{D-br}}{L}$  with L ( $r = -0.482$ ) and of  $\frac{T_{D-br}}{B}$  with B ( $r = -0.535$ ). That

means that with an increase in the figures of the diameters in the anterior view there goes a relative or in extreme instances even absolute decrease in the diameter in the lateral view (depth) and vice versa. This antagonism between the two dimensions of the heart may be considered as a special feature of the general anthropometric law of compensation.—It was found for the constitutional brachitype that  $T_{D-br}$  and L show a prevalence while B tends to be smaller; for the constitutional longitype B was found to prevail while  $T_{D-br}$  and L tend to be smaller.

2) Diameters and area The coefficient of correlation for  $T_{D-br}$  and A is 0.248 while for A and  $\frac{T_{D-br}}{A}$ ,  $r$  equals  $-0.755$ . This high inverse relation again indicates that with an increase in A the depth of the heart tends to be smaller and vice versa: relative antagonism of area as noted in the anterior view and depth, respectively.

3) Diameters, area and volume Two studies are available. One author found  $r$  for V ( $V = 0.45 \times L \times B \times T_{D-br}$ ) and B 0.950, for V and L 0.686, for V and T 0.575. Another author noted  $r$  for V ( $V = 0.63 \times A \times T_{D-hor}$ ) and B 0.51, for V and L 0.56, for V and T 0.60. However if V was correlated with the sum of T plus  $T_{D-hor}$ ,  $r$  increased to 0.82.—The shape of the silhouette, as expressed by the  $\frac{L}{B}$  ratio has no correlation with V, i.e., one may find in

different constitutional types smaller as well as larger volumes. That the same A may accompany a different depth dimension and volume ( $V = 0.63 \times A \times T_{D-hor}$ ) is shown by the following figures. In 5 healthy people (average height of 170 cm,  $\pm 3.5\%$ , average weight of 64.5 kg,  $\pm 4.6\%$ ; and average chest circumference of 92.5 cm,  $\pm 3.8\%$ ) A was 104 cm<sup>2</sup>. ( $\pm 0\%$ ), but  $T_{D-hor}$  varied from 8-10.2 cm and V from 525-669 cm<sup>3</sup>.

4) Diameters, volume and inclination The angles of inclination in the anterior and lateral view, respectively, reveal no strict parallelism. V ( $0.45 \times L \times B \times T_{D-br}$ ) likewise shows practically no correlation with the angle of inclination (lateral view).

II. Correlation of figures of cardiac silhouette and cardiac volume with certain body measurements.

1) Diameters and area The average sternovertebral diameter of the chest ( $Ch_D$ ) is 41.5% of  $Th$  (anterior view). If one arranges a large series in descending order of the  $\frac{Ch_D}{Th}$  ratio, one finds at either end cases in which  $Ch_D$  is small or large, as compared with the average figure. In the former group,  $T_D$  is found to be absolutely smaller than the average, while T and L show a relative increase. In the latter group, the ratio  $\frac{T_D}{L}$  is somewhat increased. To give an example. If  $\frac{Ch_D}{Th}$  changes from the average 41.5% to 35.5%,  $\frac{T_D}{T}$  becomes

angle of  $85^\circ$  the depth diameter was found to be on the average 70% of the sternovertebral distance.

*Lateral View.* I. Correlation between figures of cardiac silhouette and cardiac angulation.

The oblique depth diameter shows practically no correlation with the inclination of the heart, as observed in the lateral view, and its use is therefore, perhaps, to be preferred over the horizontal depth diameter, which has a correlation coefficient of 0.341.

II. Correlation of figures of cardiac silhouette with certain body measurements

a) With W and H

There is a good correlation between  $T_D$  and W. H influences  $T_D$  only if H is proportionate to W — A prediction formula for  $T + T_{D-br}$  reads:  $0.2854 \times \text{Age} - 0.4894 \times H + 1.3450 \times W + 185.84$ ;  $r$  was found to be 0.700

b) With horizontal chest diameters and computed volume of chest and trunk.

A prediction formula for  $T + T_{D-br}$  is:  $-0.1918 \times \text{Age} - 1.1737 \times H + 1.1080 \times W - 0.0706 \times \text{ant.-post. ext. diameter of chest} + 0.0784 \times \text{lat. ext. diameter of chest} + 0.7598 \times \text{girth} + 260.306$  (chest measurements taken at level of third interspace);  $r$  was found to be 0.734.

Anterior-posterior chest diameter to  $T_{D-hor}$  varied for to 2.11. It varied for to 2.11.

The correlation coefficient between  $T_{D-br}$  and an external antero-posterior chest diameter was 0.477, between  $T_{D-br}$  and an external antero-posterior hypochondrium diameter 0.401. Similarly,  $r$  was for  $T_{D-hor}$  and these diameters 0.494 and 0.504 respectively.

*Anterior and Lateral Views* I. Correlations between figures of cardiac silhouette, cardiac volume, cardiac angulation.

1) Diameters and diameters. The range of variation for  $\frac{T_D}{T}$  is considerable, it is for 9/10 of all cases, adolescent and adults, males and females 62.5% - 85.0% with an average of 73%. The corresponding figures for  $\frac{T_D}{L}$  are 55.0% - 75.0% with an average of 66.3%. Likewise the range of variation for  $\frac{T_{D-hor}}{T}$  was 60-85% for 82.6% of all the cases

If one arranges a large series in descending order of the  $\frac{T_D}{T}$  ratio, one finds at either end cases in whom  $T_D$  is small or large, as compared with the average figure. In the former group  $T$  and  $L$  are found to be absolutely greater than the average, in the latter group  $T$  and  $L$  are found to be absolutely smaller than the average. If one arranges the same series in descending order of the  $\frac{T_D}{L}$  ratio one will find again at either end cases in which  $T_D$  is small or large, as compared with average figures. In the latter group  $L$  is found to be absolutely smaller than the average. Similarly, it was found that  $T_{D-br}$  correlates poorly

disease, hyperthyroidism, anemia, hypertension, angina pectoris, well compensated valvular disease; there was no failure, either in the past or at the time of the examination. The following correlation coefficients were obtained:

Output per beat	and V	0.51
Output per minute	and A	0.43
Output per minute	and V	0.59
Work per beat	and A	0.77
Work per beat	and V	0.76
Work per minute	and A	0.69
Work per minute	and V	0.69

It follows that cardiac work, which under basal conditions is mainly determined by the product of the volume of expelled blood times the arterial resistance, is of chief interest in relation to cardiac size. (Cardiac output is of great importance in relation to basal metabolism and body size.)

2) α) Material: 31 healthy subjects. The work per beat in gram-meters was expressed by the equation  $25.7 + 0.0431 \times V$ , the standard deviation was 21.3 gram-meters

β) Material. 31 healthy subjects, 47 hospital cases without circulatory disease and 14 hypotensive cases. The work per beat in gram-meters was expressed by the equation  $15.1 + 0.0523 \times V$ , the standard deviation was 21.3 gram-meters. The output per beat in  $\text{cm}^3$  was given by the equation  $29.33 + 0.0213 \times V$ , the standard deviation was 16.9  $\text{cm}^3$ .

The following prediction formula was then derived:  $0.052 \times V - (\text{left ventricle work per beat in gr.-m}) = K$ . If K is greater than 27 the chances that there is abnormal myocardial function are about 97.5 in 100. If K is greater than 20, the chances of abnormality are 95 in 100, if greater than 12 the chances are 90 in 100.

b) Acetylene gas method 1)  $V = 0.63 \times A \times T_{D\text{-hor}}$ , teleroentgenograms, distance 200 cm. Material healthy subjects, 33 adults and 14 girls, age 10-14 years. The correlation coefficient for output per beat and V was found to be 0.84. The index  $\frac{V}{\text{stroke volume}}$  was 7.0-13.5 for 95% of all cases. This index

surpassed the normal limit in all instances of failure. 2) Kymographic method, determining the systolic and diastolic outline. The output per beat was determined from the equation  $0.67 (A_D^{145} - A_S^{145})$  wherein  $A_D$  and  $A_S$  represent the silhouette areas during ventricular diastole and systole. Since in this study the equation for the relation of A to V was  $V = 0.64 \times A^{145}$ , it follows that the net stroke output would be almost exactly equal to the maximal volume difference between systole and diastole, the total stroke output, i.e., pulmonary plus systemic circulation, would be twice this value

The ratio  $\frac{A}{\text{stroke volume}}$  has been studied for a group of girls over a period of several years of development (puberty), it remained constant.

SUMMARY. The following points deserve attention.

1) The only correct determination of heart size consists in the actual volumetric reconstruction. None of the formulae devised for computation of

67.6% instead of the average 73.0% and  $\frac{T_D}{L}$  becomes 70.9% instead of the average 66.2%. It thus can be concluded that first, a deficient or excessive development of the chest (and trunk) is associated with a corresponding decrease or increase in the depth of the heart: shallow and deep heart respectively; second, an antagonism is noted again, at least for cases deviating from the average values, between T and L (lateral-lateral development) and  $T_D$  (antero-posterior development). It was shown already in an old study, 25 years ago, that in patients with flat chests the depth of the heart is diminished correspondingly: two men with H 175 cm, W 61 and 83 kg. and an external antero-posterior chest diameter of 17.5 and 23 cm., respectively, showed A (anterior view) to be almost identical—140 and 137 cm<sup>2</sup>.; the corresponding heart depth figures (lateral view), however, were 7.5 and 12.2 cm. respectively. The antagonism between area and depth dimension of the heart can be found in the same study; in two women, each with H 164 cm. and W 58 kg., with external antero-posterior chest diameters of 15 and 20 cm., A (anterior view) was found to be 109 and 84 cm<sup>2</sup>. respectively; the corresponding figures for the depth of the heart (lateral view) were 7 and 10.7 cm. respectively.

2) Volume.  $V$  ( $0.45 \times L \times B \times T_{D-br}$ ) correlates better with W ( $r = 0.571$ ) than with H ( $r = 0.386$ ). Another investigator gave for  $V$  ( $0.63 \times A \times T_{D-hor}$ ) and W,  $r = 0.82$ , and for  $V$  and body surface,  $r = 0.84$ . These figures are unusually high, (the material consisted of 33 adults only; teleroentgenograms, distance 200 cm) —The ratio  $\frac{V}{W}$ , or the number of cm<sup>3</sup> of heart volume per kg. body weight, has been determined in three different studies, in two of which the orthodiagraphic method and the formula  $V = 0.63 \times A \times T_{D-hor}$  were used, while in the third study teleroentgenograms were taken at rectangular directions and corrections were applied for the enlargement of the heart shadow on the film with the constant factor 0.67. In the first two studies the minimal and maximal figures were 6.5, 6.7 and 11.5, 11.8, in the third study they were 7 and 13.—A prediction formula for the heart volume in males is  $V = 8W + 65$  ( $\pm 12\%$ ) and for females  $V = 5W + 150$  ( $\pm 12\%$ ).  $\frac{V}{W}$  ( $V = 0.5236 \times L \times B^2$ ) was found

for adults to be 6-10, for children 11-13 (indicating a proportionately greater heart volume).—The correlation coefficient for  $V$  ( $0.45 \times L \times B \times T_{D-br}$ ) and Cir was 0.302, for  $V$  and chest value 0.391 (here it is interesting to note that the correlation coefficient for heart weight and chest value was 0.381, as demonstrated in an extensive anatomical study), for  $V$  and trunk value 0.580.

CORRELATIONS BETWEEN CARDIAC SIZE, CARDIAC OUTPUT AND CARDIAC  
 ----- of the investigators have studied the relation of  
 the latter factors were deter-

a) Ethyl iodide method;  $V = \frac{A^{1.5}}{1000}$  ( $K = 0.53$ , omitted).

1) Material: 39 cases, normals and subjects affected by functional heart

only if the distal portion of the arch holds an orthogonal, axial direction; this is realized for the anterior view in a good number of instances. In other cases, however, this portion of the arch has a definitely oblique course. It extends far

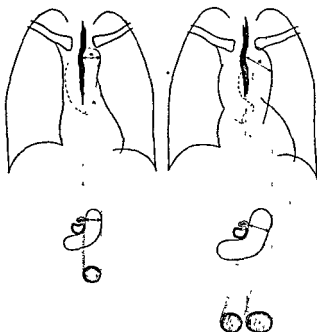


FIG. 38—Diagrammatic sketch to illustrate measurement of aortic arch (method of Kreuzfuchs) Upper part of diagram represents anterior view with esophagus outlined by means of barium paste Lower part illustrates horizontal cross-section at level of arch Left side of diagram represents normal condition, note change in course of aortic arch. proximal portion obliquely directed, distal portion has antero-posterior direction Right side of diagram represents findings with elongation and tortuosity of aorta, note change in course of aortic arch not only proximal portion but also distal portion is obliquely directed The size of aortic arch is determined by subtracting 0.2 cm (i.e. thickness of esophageal wall) from a distance which connects lateral contour of aortic knob with medial contour of esophageal lumen, as outlined by barium—distance *a*, *lt* side In instances of elongation of aortic arch, this distance gives false (i.e. too great) figures—distance *a*, *rt* side Here a correction is obtained by taking measurement with a slight degree of rotation, toward the *rt* ant obl view Courtesy Doz. Dr. E Zdansky, Vienna and G Thieme, publisher, Leipzig. From *Fortschr Geb Röntgenstr*, 1932, 45, 40

dorsad and to the left. The convexity of the aortic knob is formed by a lower segment of the aorta than that which makes contact with the esophagus. This is noted particularly in the presence of elongation or dilatation of the aorta, in association with a high position of the diaphragm or with a substernal goiter. Since the course of the second part of the arch is no longer sagittal we are deal-

the heart volume guarantees a sufficiently small error, and only those can give approximately satisfactory results which consider three dimensions of the cardiac shadow.

2) L and B should be used in addition to T or may well replace it. The  $L \times B$  product may well be used instead of A. It is necessary to consider the depth of the heart because a certain relative antagonism exists between the figures for the diameters and area in the anterior view and the diameters in the lateral view (law of compensation). This is of greater significance in those instances where a deficient or excessive constitutional development is present. It will be found for the majority of instances that cardiac silhouettes which are absolutely larger in the anterior view are absolutely larger in the lateral view also, and vice versa.

3) Considerable variation exists concerning the relation between the size of the silhouette and external somatic measurements. The correlation coefficient often stays too low to have great statistical significance. The correlation between external and internal measurements is much better manifested in the horizontal than in the longitudinal section of the body; thus a deficient or excessive antero-posterior development of the chest (trunk) and heart are rather well correlated.

4) The practical value of comparing the area of the orthodiagraphic silhouette with the size of the fist has been suggested. This correlation was found empirically and needs further statistical study; it seems to work less well for certain hearts the constitutional shape of which deviates markedly from the average.

5) Recent investigations have demonstrated a correlation between cardiac size and work of the heart; this correlation seems to be of higher statistical significance than many of the external somatic measurements and, in addition, seems to serve as a reliable measure for the functional state of the heart. Investigations are needed on a larger series of normal subjects and actual volumetric reconstruction of the individual hearts should replace computation formulae.

**Measurement of the Aortic Shadow. ARCH. Anterior View** The esophagus is visualized by means of a barium paste. At the level of the arch, the esophagus is seen to deviate to the right in a meniscus-like fashion. The contours of the barium stream and of the aortic knob may be completed to form a circle. In an anatomic cross section the sequence from right to left is: wall of the esophagus, its lumen (filled with barium), wall of the esophagus again; connective tissue, wall of the aorta, its lumen, wall of the aorta again, lung tissue. In marking, orthodiagraphically, the aortic knob and the most medially situated point along the barium contour by short vertical lines, i.e., drawing tangents to the circle, one obtains a horizontal, connecting line. This distance, when reduced by 0.3 cm., equals the diameter of this portion of the aorta. The 0.3 cm. corresponds to the size of the wall of the esophagus plus twice the size of the aortic wall, plus mediastinal pleura, plus the connective tissue that separates the aorta from the esophagus. This figure may be greater, however,

tracheo-bronchial contour is identical, or almost identical, with the actual dorsal contour of the cranial (distal) portion of the ascending aorta. But one cannot prove it. And the figures thus obtained may be too small by reason of the fact that some of the aortic and superimposed superior vena cava shadow is lost within the tracheo-bronchial air column. On the other hand, too large figures may result because of superposition of the superior vena cava shadow. The ventral landmark of measurement is undoubtedly very often represented by the ventral contour of the cranial (distal) portion of the ascending aorta. That this is not always the case has been shown convincingly by the study of the opacified aorta in the cadaver and of the calcified aorta in the living: the actual aortic contour may occasionally be farther inside (dorsad) and thus not coincide with the ventral mediastinal contour. Measurement then would result in figures that are too large.

The close relationship of the arch to the trachea permits an attempt to measure the arch in this position. Elongation and tortuosity cause much inaccuracy.

*Left Anterior Oblique View.* One notices at a certain angle of rotation and approximately 0.5-1.5 cm. below the angle of bifurcation the termination of the air column of the right main bronchus. The short line of termination, rather hazy and indeterminable in many cases, runs almost parallel to the ventral convex contour of the ascending aorta. A horizontal line is drawn to connect a point slightly above the junction of the silhouettes of ascending aorta and right heart with the vertical line of bronchial termination and has been considered to represent the diameter of the ascending aorta. The measurement may be attempted only within a very short distance. Caudally, between aorta, superior vena cava and bronchi, lies the right branch of the pulmonary artery in contact with the dorsal contour of the aorta. Cranially, at the level of the bifurcation, the aorta curves dorsad and both of its borders become invisible. Figures are said to vary from 1.9-2.9 cm. and to coincide fairly well with those which were obtained by measurement in the right anterior oblique position (see preceding criticism).

A measurement of the ascending aorta is correct only when both ventral and dorsal contours are visible. This is noted in the presence of extensive deposits of lime salts and is encountered very rarely indeed. A marked increase in contrast as noted in the higher age group and with emphysema increases the probability that the measurement will be approximately correct. In summarizing one may state that an exact measurement is usually not possible.

It has been reported that a close correlation exists between the mean of the figures for the ascending aorta plus arch and Th (width of the chest at the level of the dome of the right diaphragm). The average for the age group of 20-30 years was 10%, increasing to a maximum of 25-30% for the age groups of 46-50 years. The aforementioned criticism should be remembered.

The total width of the vascular shadow has been measured in the anterior view and 6.0 cm. was given as the upper limit. This method is altogether unreliable. The width of the aortic loop is influenced by many factors, such as the position of the diaphragm, or a but slightly scoliotic spine. In addition, the superior vena cava participates to a varying degree in the formation of the right vascular contour.



ing not with a circle but with an oval, the diameter of which is greater than the true cross section of the aorta. The point of the aorta farthest to the left is not situated exactly opposite to the junction with the esophagus but is more dorsal. The line connecting the center of the esophageal concavity with the most prominent part of the aortic knob is not horizontal but oblique and inclined either caudad or cephalad as it passes lateral. Such a measurement gives abnormally large figures. A correct measurement is still possible. One turns the patient for a few degrees into the right anterior oblique position. The orthogonal, axial direction of the distal-dorsal portion of the arch is thus regained and with it, its circular projection.

The distance from the barium curve that outlines the medial border of the aortic arch to the left border of the aortic knob, in healthy adults, may be as low as 1.8 cm. for the gracile build and as high as 3.0 cm. for an oversized individual. In the younger age group an average of 2.3-2.5 cm. is commonly found, while beyond the age of sixty 2.8-3.2 cm. is normal. In children the distance is about 0.8 cm. at the age of five and increases to about 1.4 cm. at the age of fifteen. In order to obtain the actual diameter of the aortic vessel, 0.3 cm. should be deducted from the figures obtained in adults and 0.2 cm. from the figures for children.

The measurement is done at the level of the isthmus of the aorta and this portion is known to vary considerably in size. The difference in size between the isthmus and the ascending portion has been determined anatomically and was found to vary from 0.3 to 0.8 cm. One investigator found an average ratio of 3:4, another of 4:5. The measurement is impracticable in the presence of marked scoliosis and displacement of mediastinal organs and with a disease process between the esophagus and the aorta.

A similar principle of measurement is involved when making use of the air filled tracheal lumen. A connecting line is drawn between the aortic knob and the air contour; 0.3 cm. is deducted, accounting for the size of the wall of the trachea plus twice the size of the aortic wall, plus mediastinal pleura, plus the connective tissue that separates the aorta from the trachea. As compared with the method which makes use of the aortic arch impression on the esophagus one finds the figures to be less correct. This is accounted for by the presence of

vertical diam-  
eter level of the

summit of the aortic arch shadow and if its caudal end is allowed to coincide well with the caudal margin of the aortic arch.

**ASCENDING PORTION (ATTEMPTS).** Attention is directed to the detailed roentgenologic-anatomic description in chapter III.

**Right Anterior Oblique View.** At about 35-40° the clear contour between

of the ascending aorta. Many authors have advised measurement graphically at a level halfway between the aortic arch and the pulmonary conus. The normal figures are said to vary from 1.8 to 3.0 cm., mainly depending upon age. The criticism of this measurement can be shortly summarized as follows: it seems possible that in a large number of instances the ventral

A heart with a T of 15 cm., one third to the right and two thirds to the left, with its main axis 10 cm. away from the film shows, at a distance of 200 cm. a magnification of 0.86 cm. = 5.7%; at a distance of 300 cm. this is reduced to one-half. For an infant with a T of 6 cm the magnification for 200 cm was found to be 0.07 cm., for a child with a T of 10 cm, 0.19 cm.

In order to correct the distortion due to divergence of the rays, a correction factor should be determined; it is always  $<1$ . It is derived from the target-film distance, distance of the center of the heart from the ventral chest wall, (vide supra), and distance of the ventral, bony-cartilaginous chest wall from the film.

In order to obtain for the same heart size but with different heart-film distances a constant magnification of 5%, a different target-film distance must be employed. Example: for a heart-film distance of 5.9 cm.—124 cm, for 9.15 cm.—191 cm. and for 13.7 cm.—289 cm. The magnification for the lateral chest contours, as compared with the heart contours, is necessarily increased. The magnification of the area of a normal sized heart silhouette, the center of which is considered to be 10 cm. distant from the film, and for a target film distance of 200 cm, amounts to about 10%. The degree of magnification is increased in the oblique and lateral views because of the increased object-film distance; and it is increased in the event of enlargement of the cardiovascular shadow, partly, because the points along the silhouette are farther away from the mid-line of projection and, partly, because their distances from the film are increased.

*Orthodiagraphy* represents a parallel projection. It permits of a greater degree of completion for the caudal heart border and an exact determination of the junction between atria and ventricles; ventricles and great vessels; and among the great vessels. It is of inestimable teaching value in that it necessitates the completion of the fluoroscopic observation and impression by an objective drawing; it is inexpensive. It requires a technic which must be mastered thoroughly and requires great accuracy for its application, for instance, to err 2 cm in centering, will involve an error of 4 mm. for each heart border. Nevertheless the personal equation is unavoidable, orthodiagrams from different institutions are usually different as to the shape of the single arches and the distinctness with which the junctions are marked. *Teleroentgenography*, within the commonly used distances, is a central projection; it records by photographic means complete detail and charts, as landmarks, all bony structures of the chest, it involves some expense and requires for special purposes a careful technique.

It is usually believed that teleroentgenography is the more accurate procedure; in a certain sense, however, this is a false assumption. If one compares separate, carefully made orthodiagrams with various routine teleroentgenograms, both made of the same patient, it will be found, as a rule, that the numerical differences are greater in the teleroentgenograms. The greater error in the teleroentgenogram lies partly with differences in projection and partly with differences in the respiratory and cardiac phases.

**Criteria of Comparison.** While orthodiagrams are acceptable as records of a single examination, they cannot be accepted for purposes of comparison, unless the changes of shape and size are very marked or unless a very special

**Pulmonary Artery Branch.** The large comma or sickle-like right lower branch is well visualized in the anterior view and even better with a slight degree of rotation into the right anterior oblique view. It is situated in the immediate vicinity of and lateral to the right main bronchus, and their borders are almost parallel for a distance of several cm. Here the measurement is taken. The average figure for the diameter is 1.0 cm.

**Distance of Silhouette from Anterior Plane.** The distance of the farthest lateral points of the cardiovascular silhouette from the film and skin level respectively may be determined by any of the methods which are used for foreign body determination. The average skin distance for the right border

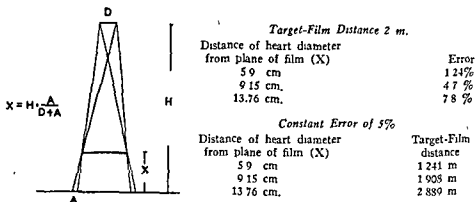


FIG. 39—Magnification in Teleroentgenograms. If two exposures of several seconds duration are taken at the target-film distance  $H$  and by stereoscopic shift (distance  $D$ , 1 cm per 10 cm target-film distance), the two resulting respective cardiac borders will be separated by the distance  $A$ .  $x$  is the distance of the main diameter of the heart from the plane of projection and can be determined from the known factors  $H$ ,  $A$ ,  $D$ . The degree of magnification depends, of course, upon the distance  $x$ . The respective errors for 2 m., due to magnification, expressed in %, are given in the upper half of the table. Three examples are chosen. In order to obtain a constant error of magnification of 5%, the target-film distance would have to be changed for different values of  $x$ , this is given in the lower half of the table. Adapted from Haret and Frain, *Bull. Mém. Soc. Radiol.*, 1932, 20, 35.

is 4-6 cm., for the left lower pole region 3-4.5 cm. for the ascending aorta 5-7 cm., and for the pulmonary artery 5 cm.

**Magnification in Teleroentgenograms and Comparison of Orthodiagrams with Teleroentgenograms.** Ordinarily teleroentgenograms are obtained by central projection. The degree of magnification is determined by the target-film and object-film distance, and by the size of the object and its position in relation to the central ray. The main axis or the center of the heart is approximately at the border of the ventral and middle third of the antero-posterior chest diameter. The depth of the most lateral points of the cardiovascular silhouette is determined by any of the methods used for the determination of foreign bodies.

The results which are obtained by actually comparing orthodiagrams and teleroentgenograms of the patient are far from satisfactory; the reason is that the cardiac phase, phase of respiration, and body position are seldom identical

technique is used (vide infra). The important problem in comparison of records, taken of the same patient at different times, necessitates discussion of certain standards for that comparison; the errors commonly committed; and the methods of recognizing and avoiding them

1) The target-film distance must be maintained inviolate. Changes of  $\pm 10$  cm. are negligible for standard distances in excess of 180 cm. The identity of this distance is recognized by measuring the width of the chest. It is necessary that the respiratory phase be identical and that practically no weight variations occur.

2) The target, the midline of the patient and the midline of the film must be centered. The deviations must not exceed 1-2 cm. for the tube and 3-4 cm. for the patient. a) A vertical lead mark a few cm. in length should be attached to the top of the film holder, indicating the midline and a similar lead mark be fixed along several of the upper thoracic spinous processes. The shadows of these vertical marks must coincide in the various films. b) A horizontal lead mark, a few cm. in length, is held by an arm which is fixed to the frame and moves with the film holder; its distance, from the tube, is approximately 30 cm and the height of the arm is one-half the vertical length of the film holder (film). A similar mark is attached to the film holder on the same side. The shadows of these two horizontal marks must coincide in the several films. Vertical deviations of the tube will cause a different projection of the heart and diaphragmatic contours in relation to the intercostal spaces, it will therefore not be possible to judge as to whether the diaphragmatic position has actually changed or not. Changes in the vertical position of the chest are usually slight; however, special consideration is to be given to this point in growing children.

3) The chest must not undergo any change in position either in the sense of a) rotation along its long axis, or b) increased lordosis or kyphosis. The best method for recognizing such alterations is a careful study of the bony skeleton of the chest. The ventral and dorsal portions of the ribs show a network pattern on the film with small, differentiated and characteristic meshes which are symmetrical for identical rib crossings at either side, provided the chest is symmetrically built. Any change in the position of the chest is recognized by a variation in the shape of the rib pattern. Such changes are best studied by sketching the rib pattern of each film on transparent paper or film base, with superposition of these copies. Films are comparable only if two such tracings are as identical as are two superimposed prints of one and the same film. Rotations are especially likely to cause distortion of the vascular shadow. In order to avoid too great alteration in the chest position, the use of a binder is advisable.

4) The position of the diaphragm must be identical; it is determined by relating the dome of the right leaf to the last visible dorsal rib or intercostal space. The respiratory phase and the degree of the abdominal content are to be reckoned with. The same respiratory phase is guaranteed either by using long time exposures which include several cycles of the respiration or by making use of the technical devices referred to in chapter I. Deep inspiration is to be avoided as previously indicated. At the time of the follow up study, the diaphragm may be found in a position either farther cranial or caudad with

FIGS 40, 41, 42, 43, 44 — 9 yrs, F Active rheumatic carditis with slight degree mitral valvular lesion. Serial roentgen ray pictures were taken to determine possible changes in size and shape of heart. Two such films, taken at different times, are shown. Silhouettes appear different and these findings were thought by a clinician to be indicative of plastic pericarditis. A careful analysis, however, shows that the two films, both taken at a standard distance, are not comparable. The rib patterns were redrawn on filmbase, each photographed and, in addition, superimposed. The lack of coincidence is obvious. The reason for it is a difference in the position of the trunk which was slightly kyphotic and lordotic, respectively. See "criteria of comparison," chapter IV.



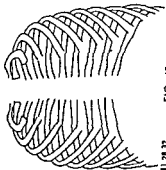
FIG 41

FIG 40



FIG 43

1-18-34



1-18-32

FIG 42

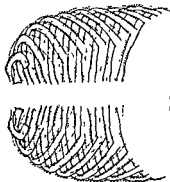


FIG. 44

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changes in body weight usually responsible for it. In the former case, a lavative may be employed; in the latter, an elastic bandage around the abdomen. When cardiac failure subsides, lowering of the diaphragm follows a shrinking liver and reduced abdominal distension, and the position of the heart changes. Comparison of heart size in these instances becomes practically impossible.

5) The phase of the cardiac cycle must be identical. This is obtained either by using exposures of longer duration and thus procuring the largest possible cardiovascular silhouette for a given view and distance, or by taking exposures at a determined phase, as discussed in chapter I.

The following detailed technique has been used to gain comparable orthodiagraphic records. a) a narrow strip of lead is applied along the spine, from the sixth cervical to the eighth thoracic vertebra. a wire is fixed down the anterior midline from the jugular notch to the region of the umbilicus, cross wires are placed across the sternal angle and also across the sternum at the level of the fourth intercostal space. Finally, small wires are placed over each nipple and the maximal apex beat. b) a wire is strung along the center of the table; two others are placed at right angles to the former, 60 and 70 cm. respectively from the head-end of the table. The patient is examined in the recumbent position so that the respective longitudinal lead stripes, wires and upper cross wires are superimposed with the lower cross wires in parallel.

Statements referring to changes in the size of the cardiovascular silhouette are acceptable only when careful criteria are fulfilled. Recourse to the majority of publications upon this subject reveals much evidence of vast waste of both energy and material, with concomitant confusion and contradictory conclusions.

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ly determine the shape or size of the structure producing it and therefore is not a reliable basis for estimating the movement of the structure. The pulsatory changes occur in a three-dimensional space while the registration and observation of these pulsations take place in one plane. In no position is it possible to observe or register the activity of all four heart chambers simultaneously, and linear or area changes in the sense of increase or decrease, as observed in one position, may be well compensated in another

5) The pericardium covers the whole heart and the roots of the large vessels; it is fixed along the lung roots, diaphragm, inferior vena cava and to some degree in the anterior mediastinum. It cannot therefore follow the movements of the heart. Strictly speaking, we register roentgenologically deformations of the pericardial surface caused by the underlying heart action and extrapericardial vascular movements.

6) Along the surface of the mediastinum, with the exception of a small frontal area, a pressure is sustained which is expressed by the atmospheric pressure minus the traction exerted by the elasticity of the lungs which are distended beyond their equilibrium. This traction or suction effect is said to amount to several kilograms and is multiplied about three times during forced inspiration. It is increased during the Müller test and decreased during the Valsalva test and also when the elastic property of the lung tissue diminishes (emphysema, collapse). This suction effect becomes affectual during the ventricular systolic diminution of heart size and presumably prevents the thin-walled atria from collapsing at that time.

7) The roentgenologic medial movement of the left lower cardiac contour is usually identified with systole; this is only partly correct. Ventricular systole starts with a short period of rising tension. This isometric contraction is apparently associated with some degree of rotation and change of shape of the heart. The "roentgenologic" systole, in the conventional sense, obviously indicates the period of ventricular emptying. It has been demonstrated that during early systole, when the apex and base are moving rapidly toward each other, the left ventricular border is actually moving outward instead of inward, and that this movement is followed and continued, to be sure, by the relatively slow inward movement. Therefore in studying a graph that depicts the movement of the lateral wall, one cannot quite identify it with a ventricular volume curve, notwithstanding their resemblance.

It must be realized that the pulsatory movements of some structures are normally not visible. These are that part of the ventral heart border which is in the immediate vicinity of the sternum; the atrio-ventricular septum including the movement of the heart valves, the first part of the ascending aorta in its total circumference, the mesial contours of the superior vena cava, the ascending portion and arch of the aorta and pulmonary artery, the greater part of the caudal heart border where it fuses with the liver shadow. It is also difficult to see pulsations in the left lower pole region in the presence of a well-developed transverse pericardial shadow.

The motion of the ventricular septum is of two types: 1) a centripetal and centrifugal movement at the margin which is

## CHAPTER V

### DYNAMICS OF THE CARDIOVASCULAR SYSTEM

**General Remarks.** It is important to bear in mind the following facts whenever one undertakes the roentgenological observation and study of cardiovascular pulsations

1) The fluoroscopic and graphic registration of these pulsations is practically always undertaken with central projection (divergent rays). The actual amplitude of movements is increased, depending upon the target-screen (film), target-object and object-screen (film) distance. While parallel projection delineates the most laterally distant points of an object, central projection strikes tangentially the more posteriorly and centrally located points.

2) The actual amplitude of a movement is fully registered only when its direction is at right angles to the direction of the central ray. As the direction of the movement approaches that of the ray projection, the projection of the amplitude correspondingly diminishes. The movement of any point in question is the result of various components, none of which can be exactly determined, and it is practically impossible to draw conclusions as to the magnitude of but one of these components—in this case that of pulsation. There are, in addition, two other complicating factors: a) One cannot determine the actual size of the lateral movement of the heart wall because the point in question would have to move exclusively parallel to the plane of observation and exactly longitudinal in the slot used for kymographic registration, i.e., at right angle to the direction of motion of the film. b) Lack of movement does not indicate that the underlying part did not pulsate since displacement opposite to the direction of pulsation may have had a counteracting effect. For instance, a point at the surface of the left ventricle changes its position during systole on account of the following movements: centripetal (decrease in the heart volume), centrifugal (increase in the curvature of the surface), ventral and cranial (lever-like displacement), ventral and mesial (torsion of the heart). To these intrinsic changes in volume, shape and position may be added the influence of respiration. Thus any given point on the cardiac silhouette may at successive moments correspond to different anatomical points.

3) The following experiment shows that the pendulating and rotary motion of the heart as a whole is so well marked during systole as to entirely obscure any possible systolic contraction of the outer contours of the ventricular wall. Metallic indicators were inserted at definite spots in the wall of the left ventricle in sheep. The paths of these indicators were recorded by roentgen cinematography, and E.C.G.'s were taken simultaneously. Screen pictures were cinematographed in five different directions. The paths of the indicators as shown on the films were graphically recorded, picture by picture, to obtain 1) an idea of the spatial paths of the various indicators, 2) a time comparison with the E.C.G.

4) A study of the movements of the silhouette border yields information regarding the movement of shadows. But the shadow does not characteristical-

10 cm in contrast to the average reading distance of 38 to 40 cm. A refraction should be had prior to fluoroscopic work to insure maximum visual acuity. In the group with normal accommodation for their age it should be taken into consideration that, if their accommodation near point is greater than 10 cm, compensation should be made by finding the lense suitable for this work. The table for normal accommodation for the different ages can be found in any standard textbook of ophthalmology. In the presbyopic group bifocal addition should be strong enough to effect a clear focus at 10 cm. A rough approximation for the completely presbyopic group is a plus 8 diopter addition.

**DARK ADAPTATION OF THE EYE** Intensity discrimination and visual acuity, corresponding roughly to contrast and sharpness in roentgenograms, deteriorate as a result of lowering of the brightness level. The retinal elements concerned with vision are the rods and cones. Both contain pigments. Vision at high illuminations is color vision and is most efficiently carried on by the cones in the center of the retina, while vision at low intensities of illumination is colorless and is essentially carried on by the rods in the periphery of the retina. On an abrupt change from light to darkness the visual acuity diminishes while there is an increase in the light sensibility of the peripheral portion of the retina. This quantitative increase in the retinal sensitivity is designated as dark adaptation. It occurs in two parts. The first begins at once, is due to cone function, is over in 3 or 4 minutes, and the increase in sensitivity is approximately ten-fold. The second part begins somewhat later, is due to rod function and increases in the form of a logarithmic curve, the knee of which is reached after 20 to 30 minutes or more. After a 20 minute interval the increase in sensitivity is at least 1000-fold and it continues to increase for many hours, though not in comparable degrees. Dark adaptation takes a shorter time if the transition is made from artificial light instead of daylight—particularly sunlight. Hemeralopia or night blindness, which is failure to see clearly in dim light, is due to vitamin A deficiency. This vitamin is an essential constituent of the retinal pigments, and the effect of an inadequate supply is to slow down the rate of regeneration of these pigments. Active vitamin A is available in the butterfat of milk, the yolk of eggs, in fish liver oils and, to a lesser degree, in certain animal fats. Provitamin A occurs in carrots and green leaves. Vitamin A adequacy is not only governed by intake but also by absorption, and by the conversion mechanism of provitamin A into the active form. Hemeralopia is particularly found in patients with liver damage that is most outspoken in cirrhosis of the liver, with a marked degree of thyrotoxicosis, with diabetes, or

paratively large diameter in the dark tends to require less time for dark adaptation. It is well to remember that the pupil diameter in the dark decreases with age. Retinal illumination is of course reduced if the pupils are miotic and

to describe the normal will merely give an average picture

*Anterior View.* The left lower border shows alternately a rapid inward



roentgenologically distinctly recognized as a pulsatory movement. It is important to bear in mind that the longitudinal component is far greater than the movement that appears at the margin. The following proofs support this statement. 1) in animals, if lipiodol is injected into the pericardial cavity of rabbits, it gathers principally in the atrio-ventricular groove; its movement, as registered by kymography, amounts to  $\frac{1}{3}$ - $\frac{1}{4}$  of the total length of the heart while the border of the silhouette, including the apical portion, moves but little. If barbed points of a fish hook or bits of silver wire are placed near the atrio-ventricular groove, these movements are seen to be far greater than the undulations of the ventricular border. 2) in man. a) whole bullets or fragments were observed to be located within the heart near the atrio-ventricular septum. The amount of excursion was 1-3 cm, which is much more than the visible outer pulsations. Calcareous deposits in this septum can be observed to move

The patient was placed in such a position of recumbency that both the movements fell into the horizontal slit of the roentgenkymograph. The corresponding movement of the left ventricular border was small in comparison with the degree of longitudinal shortening. b) one may consider the left ventricle as a paraboloid with a height of 10 cm. and having as a base (mitral ring) a circle with a radius of 3.5 cm. If one now assumes that all parts, with the exception of the base, move medially a distance which corresponds to the visible pulsations and if one computes the difference in volume one obtains a figure which is far below the figure for the normal stroke volume, therefore an extensive movement of the base must be present. c) a wax model of both ventricles representing a volume of about 400 cm<sup>3</sup>. may be constructed. The normal stroke volume of 120 cm<sup>3</sup>. is not obtained by subtracting from all surfaces the equivalent of the normal outer roentgenological amplitude. It is necessary to remove an additional layer of 2.5 cm. thickness from the base.

A differentiation of the pulsations of single heart cavities is undoubtedly made difficult on account of the fact that the pericardium bridges over and smooths out separating grooves.

**Fluoroscopy.** Information is improved by the following factors: good contrast, the presence of a slower heart rate, increased distance between the fluorescent screen and chest wall of the patient which magnifies all movements, alternate use of a smaller and a wider aperture of the shutter, and slight to-and-fro rotations of the patient. Small objects are observed more readily when they are in motion. The comparison of the improvement of the visibility of small objects as a result of motion over that obtained by increasing the current strength, made it possible to establish a quantitative estimate of this improvement. It approximates 10-15%. On account of the rather poor

very short distance; it  
the right and left side  
to be a help. A fine-grid

Potter-Bucky diaphragm is useful in improving contrast

**ACCOMMODATION OF THE EYE.** It should be realized that when studying details of the fluoroscopic image the observation distance may be as close as

rather indefinite short movement which sometimes appears to precede slightly the movement caudal to it. While the left lower contour moves medial, this area shows a short caudal displacement.

The *right* lower border has an amplitude of about 0.2-0.3 cm. and shows different types of movements. It may move in a lateral direction while the left lower contour moves mesial; it may have, especially at a low heart rate, a medial direction so that the whole heart shadow contracts in a concentric fashion; sometimes the cranial third starts with the medial movement, and this

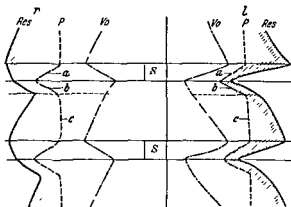


FIG. 46.—Schematic drawing of kymographic movements of lt and rt heart borders for an average case, showing interference of marginal movements, expressing volumetric change and positional change (pendulum-like swing). The shaded line (*Res*) represents the actual resultant graph as a summation effect of the two movements, which are represented graphically by the dot-dash line, *Vo* (volumetric change), and by the broken line, *P* (positional change). *S*, expulsion period. Note that the trough at rt side is reached later than on lt side. If the pendulum movement would exceed the marginal volumetric movement, small peaks would appear in the graph on the rt side during the expulsion period, and these may be interpreted erroneously as evidence for a true atrial movement. Courtesy Dr K Heckmann, formerly of Munich, now of Istanbul, and J Springer, publisher, Berlin. From *Ergebn inn Med*, 1937, 52, 569.

is followed by the caudal two thirds, a double stroke medial movement is likewise observed; and finally there may be a short rapid medial movement, followed by a movement in the opposite direction. With the vertical small heart the cranial portion may show a definite, systolic lateral movement. Marked rotation of the heart including the conus arteriosus, probably in connection with an abnormal narrowness of the right atrium, may account for this; or it may be merely an expression of a marked pendulum movement. The caudal heart border is to some extent visible under the following circumstances: (a) with the presence of air in the upper pole of the stomach, (b) in the vertical heart; (c) during forced inspiration; and (d) while the examination is made in a marked lordotic position and/or when the position of the roentgen ray tube is lowered. The whole caudal heart border is visible if gas or air is present between the surface of the liver and the diaphragm. This is the case with interposition of the colon and with pneumoperitoneum; the heart appears in a more transverse position and one notices between the right and the middle

movement, and a slower outward movement. The inward movement seems to vary in speed for different people; it usually has the appearance of a superficial, wave-like motion which progresses from the base toward the lower pole. The greatest amplitude, varying from 0.2-0.5 cm., is found, in the upright position, to be usually about 1.5-2.0 cm. above the lower pole; however, there may be no gross preponderance at any one region. In the recumbent and right lateral positions the maximum is found nearer to the base, about half-way along the arch. In the left lateral position, the maximum is near the lower

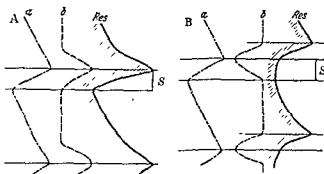


FIG. 45—Schematic drawing of kymographic movements of the cardiac border for two areas, A, corresponding to apical portion, and B, corresponding to basal portion. It is assumed for A, an area near the apical portion, that there is a summation effect of the marginal movements due to volumetric changes, dot-dash line (a), and due to systolic change in shape, broken line (b). The shaded line represents the resultant graph (Res). It is assumed for B, an area near the basal portion, that there is an opposite effect of the marginal movements due to volumetric changes, dot-dash line (a), and due to systolic change in shape, broken line (b). The shaded line represents resultant graph (Res). S, expulsion period of the cardiac cycle. When comparing B, which represents a movement at the base of the left contour, and A, which represents a movement near the apical portion of the left contour, it is noted that the inward movement at the base precedes the inward movement at the apex. This leaves one with the impression of a rather slow, peristaltic-like movement which, of course, does not exist since the excitation of the basal and apical portions of the ventricles takes place in a minimal fraction of a second so that the resulting contraction must be observed by the eye to be simultaneous. Hence an interference of volumetric changes and of changes in shape may result in a type of movement which simulates a peristaltic movement. Courtesy Dr. K. Heckmann, formerly of Munich, now of Istanbul, and J. Springer, publisher, Berlin. From *Ergebnisse inn. Med.*, 1937, 52, 576.

pole. With the vertical type of heart, the maximum is found to be at the lower pole. With the transverse type of heart, the maximum is found to be at the upper pole. With the oblique type of heart, the maximum is found to be at the lower pole. A decrease in the heart rate. By turning the patient slightly into the oblique positions it is noted that the maximum of the amplitude shifts. In the left anterior oblique view it moves toward the lower pole; for the transverse type of heart the necessary angle of rotation is larger. In the right anterior oblique view it moves toward the base and finally disappears. As compared with the average oblique type, the angle at which this movement disappears is greater for the vertical and smaller for the transverse type. Near the cranial portion of the left lower border, there is observed either an area of quiescence or a

the outline is observed. Craniad an indentation is often observed above which the pulsations are less vigorous and different in time. The level of this indentation changes during the cardiac cycle. We are obviously dealing with the left atrioventricular border. Near the diaphragmatic border, or just above it, one can occasionally observe another indentation, best seen during systole and in vertical hearts; this is another point of relatively little motion, a kind of groove. It most probably represents the juncture of the ventricular walls with the caudal end of the interventricular septum. As far as it is visible the caudal contour shows mainly a vertically directed movement. In front, the pulsations are much smaller; at a certain angle one may observe a time difference between a longer lower and shorter upper portion.

With a sufficient degree of angulation, the cranial-ventral contour (ascending aorta) shows well marked movements. Pulsations are often though not always visible along the cranial contour of the aortic arch, dorsad to the trachea, and along the dorsal contour of the very first portion of the descending aorta; the rapidity and size of the excursions as well as the special conditions of contrast play a rôle. The inner contour of the aorta is not visible and therefore does not reveal pulsations. Within the clear space of the aortic window a faint movement of the edge of a cranial-convex shadow may be seen; it is the pulsating left main branch of the pulmonary artery.

Children show comparatively large excursions in proportion to the heart size.

Kymographic studies have shown that over long periods the pulsations remain constant in extent and nature for a given individual and for any particular region of the cardiac contour, provided the physiologic conditions remain unchanged.

All pulsations are increased following exercise.

**Graphic Registration. ROENTGENOGRAPHY.** The study of instantaneous roentgenograms, taken at determined points of the cardiac cycle, has shown the following findings for typical cases. In the anterior view and at the time of ventricular contraction, the left lower contour moves mesiad, the lower pole mesiad and craniad, simultaneously the right lower contour moves laterad. The left auriculoventricular junction may be clearly seen to be displaced caudad during a period which corresponds in the electrocardiogram to the distance from the end of the R to the end of the T wave. Sometimes, however, this subdivision cannot be visualized and the second pulmonic contour simply continues into the third left ventricular contour. The cardiac shadow as a whole takes on a more globular form. In the left anterior oblique view the indentation between heart and aortic contour in front descends toward the end of systole and ascends toward the end of diastole. When correlated with the electrocardiogram, the cardiac shadow is found in this view to present its largest projection at the time of the Q-R interval, i.e., at the very beginning of the electric systole and preceding the mechanical systole of the ventricles.

**CINEMATOGRAPHY.** Only a few normal cases have been studied in detail. For the anterior view the cranial and caudal parts of the left lower contour are found not to move simultaneously during systole, the former moves laterad  $2/15$  second after the latter has begun its diastolic excursion. The caudal portion of the right lower contour moves mesiad simultaneously with the left

thirds of the caudal heart border a change in the type of pulsations, indicating the intersection between the right atrium and the right ventricle

The direction of pulsations of the caudal heart border, if visible, is craniad during systole.

The amplitude tends to decrease during inspiration, especially in people with a large chest capacity and powerful inspiratory muscles, and to increase during expiration

The great vessel borders on the left side show a rather quick lateral and cranial movement which takes place almost simultaneously with the medial movement of the left lower cardiac border. Thus a kind of see-saw action is observed. The amplitude at the aortic knob averages 0.2 cm. and when different cases are studied one finds much less variation as compared with the respective left lower ventricular contours. At the right there may be hardly any movement at all, especially when the contour appears quite straight; within the great vessel shadow there may be noted occasional and barely visible pulsations (aorta); or the whole right contour may pulsate laterally, simultaneously with the pulsations at the left side; this is well marked when the contour is convex. Or the straight contour may, especially in the presence of a high diaphragm or in the recumbent position, present a faint wave-like motion; and finally the caudal and cranial contours may show slight differences. In a word, we are dealing with pulsations of the aorta, transmitted or direct, and of the superior vena cava, and combinations of them. The aortic arch, below the

able change it these pulsations by no means express changes in the lumen only but that a pulsatory change in the position of the vessel exists.

The intrapulmonary vessels in either hilar region may show slight pulsations, especially in cases with a slow heart rate, with vigorous heart action, and in children. Contrast is an influencing factor. Pulsations are best observed at a certain degree of rotation, slight rotation for the right hilus; more for the left

*Right Anterior Oblique View* Both ventral and dorsal pulsations are quite small. The cranial portion of the ventral cardiac contour (conus pulmonalis) and the ventral and cranial contour of the visible portion of the aorta reveal more pronounced movements. The dorsal contour of the ascending aorta does not show any movement, neither does the ventral contour of the descending aorta, if visible at a higher degree of rotation. The caudal cardiac contour has vertically directed movements. In turning toward the right lateral position, marked pulsations suddenly appear along the caudal-dorsal aspect of the cardiac shadow, i.e., the left ventricular area is visualized again.

*Left Anterior Oblique View.* One notices the maximum of pulsations dorsally, even taking into consideration the increased magnification by central projection. The farther away from the screen and the pulsations are view. In its caudal portion, just ination of a ventral and dorsal with a cranial and caudal movement, and an almost peristaltic-like change in

often show the following details. Corresponding to the beginning of the ascending limb (diastole), there is noted a medial ventricular movement which flattens out, then follows a slight rise, a sudden peak and a fall (atrial systole). Corresponding to the peak of the ventricular graph at the left side there occurs a sudden, slight lateral movement; this is either due to a volumetric change or represents a displacement of the right atrium during the pre-sphygmic ventricular period. While the ventricular limb descends (systole), the atrial graph often shows a flat descent, a movement toward the mid-line, in spite of the fact that this is the period of atrial filling. This medial movement of the ventricular type is especially pronounced at the right heart border near the diaphragm. It has been interpreted as a proof that the right ventricle is forming a part of the lower right heart border but this assumption is not conclusive.

During deep, forced inspiration one often notices the following changes. The graph corresponding to the left lower contour shows very little or no change of the base line, while the height of the diastolic excursion decreases. The graph of the right lower contour shows that the descent during ventricular diastole is much flatter, and during ventricular systole there is a rise, or outward movement. The former finding may perhaps be interpreted as interference with atrial emptying, the latter, as being caused by increased influx of blood. These changes just described and observed on each side, explain, at least in part, the pendulum movement



FIG 48—Healthy individual Kymographic tracings lt and rt cardiac borders. Film moved from above downward. Upper line—beginning of ventricular systole (lt), closure time of a-v valves (rt). Lower line—beginning of ventricular diastole (lt), opening of a-v valves and beginning of atrial emptying (rt). In this instance, movements of both cardiac borders occur mainly in the opposite direction. During fluoroscopy there results a pumping type of cardiac action. Courtesy Doz Dr E Zdansky, Vienna and G Thieme, publisher, Leipzig. From *Fortschr Geb Röntgenstr*, 1933, 47, 648.

Tracings of the region of the aortic knob show the typical arterial graph, a short steep rise and a longer oblique descent. The right vascular contour may be represented by tracings which contain all details of a phlebogram; in other instances the tracing is indistinct; and again in others, aortic pulsations are registered.

**Multiple Slit** It has been demonstrated for the anterior view that the

lower pole while the movements of the cranial portion are simultaneous with the left auricular region.

**KYMOGRAPHY.** *Single Slit.* A typical tracing of the *left* lower contour, in the anterior view, has the following appearance. It consists first of one rather long and rather oblique ascending branch. This corresponds to the diastolic



FIG 47—Healthy individual Kymographic tracings of *lt* and *rt* cardiac borders. Film moved from above downward. Upper line beginning of ventricular systole (*lt*), closure time of a-v valves (*rt*). Middle line, beginning of ventricular diastole (*lt*), opening of a-v valves and beginning of diastolic atrial emptying (*rt*). Lower line, additional rise in diastolic ventricular filling (*lt*), due to atrial systole (*rt*). In this instance, movements of both cardiac borders occur mainly in the same direction. During fluoroscopy a pendulum type of cardiac action results. Courtesy Doz Dr E Zdansky, Vienna and G Thieme, publisher, Leipzig. From *Fortschr. Geb. Röntgenstr.*, 1933, 47, 648.

filling. In the beginning it rises with a steeper portion, expressing the sudden blood flow into the ventricle; this is followed by a flat part which ends with a short rise, registering the additional inflow of blood, caused by the atrial systole; but the greatest volume is almost reached when atrial systole takes place. The ascending and descending branches are separated by a rounded peak. At this turning point a small positive wave may be seen which, when correlated with the electrocardiogram, corresponds to the end of the T wave. The descending branch is shorter and steeper. It corresponds to the systolic emptying. Such tracings for different normal hearts are fundamentally similar.

A certain point in the kymographic curve will always correspond to a certain point in a simultaneously made electrocardiogram, provided the same point at the silhouette is chosen. When kymograms are taken from different points, for instance along the left lower silhouette contour, identical points in the different kymographic curves will not, however, quite correspond to identical

points in the respective electrocardiograms.

Tracings of the *right* lower contour show marked individual differences. In comparing the graphs of both heart contours, one may find that they are roughly similar so that they enclose a band which has a zig-zag form; this obviously corresponds to the type of pendulum movement which can occasionally be easily observed by fluoroscopy. The main branches of the two graphs may also have an opposite direction, in relation to the midline; the enclosing band appears alternately smaller and larger, this obviously corresponds to the type of concentric narrowing and widening which is readily observed in fluoroscopy. The analysis of the graph of the right contour will

medially-laterally but also proximally during the systole and distally during the diastole. A small peak or serration appears in the trough at the beginning of the diastolic limb. This should be correlated with the aortic valve closure and is particularly well noted in hypertensives. This finding expresses a jerk-like shift of the heart to the left, and is caused by the impact of blood in the aorta and a rebound resulting therefrom. It was interesting to find that maximum inspiration increases the caudal amplitudes while maximum expiration increases the cranial amplitudes. In the left anterior oblique view, a large diastolic ventricular excursion can be demonstrated. The *right* lower contour in the anterior view again shows either an aortic or ventricular or a mixed type of

single slit kymographic records do not support this statement. The region of the left auricular appendage yields the following various tracings: a descent, preceding the presphygmic period of the ventricle; a descent together with left ventricular contraction, a steep rise together with the top of the aortic and pulmonic tracing; or an elevation and decrease at the time of valvular closure. Obviously it is influenced by the adjacent heart chambers and large vessels. The point of quiescence, at the border of the left lower contour and region of the left auricular appendage or base of the pulmonary artery, moves slightly caudad during systole and slightly cranial during diastole.

The graphs of the pulmonary artery shadow show a flattened peak, those of the aortic shadow a sharp lateral thrust and a slowly descending limb which is broken by a notch, this depicts the closure of the semilunar valves. The movement of the medial aortic arch border can be ascertained after the esophagus is outlined with barium paste. When comparing this movement with the one observed along the lateral arch contour (aortic knob) one finds that the direction of pulsation is the same and the amplitudes almost identical *in degree, i.e., the change in position which the vessel undergoes greatly exceeds the actual volumetric change.* The amplitude of the movement is foreshortened by projection because it can be demonstrated in the oblique views that the arch moves predominantly in the ventro-dorsal and dorso-ventral direction, respectively. When studying the shadow of the cranial portion of the ascending aorta in the right anterior oblique view, one finds some discontinuity in the total movement. This again makes it very probable that a displacement of the aorta as a whole is an important component in the visible aortic pulsations. There is no close relation between the amplitudes of aortic and left ventricular borders. Variations, from case to case, in the elastic property and vasomotor innervation of the aortic wall, and in the status of blood pressure and pulse pressure, may account for such discrepancy. The following concept has recently been suggested for the aortic type of movement of the right vascular border, observable in the anterior view. It is a summative movement due to an aortic as well as to a transmitted ventricular component. The aortic component itself consists of a true widening of the vessel plus a pulsatory change in position. As the ventricles fill, the medially directed movement of the ascending aorta changes into a slow lateral movement. During the moment of isometric contraction, the ventricles suddenly become erect, which causes a quicker lateral movement of the aorta and this is sustained by the rise



different portions of the *left* lower contour do not reach the maximum outer or minimum inner contour at the same time. The medial movement commences somewhat later in the caudal parts. One author found a time difference of about 0.08 sec., others 0.1-0.28 sec. (for a cardiac cycle of 0.7-0.9 sec.) On the other hand the lateral movement very frequently begins earlier in the caudal parts. To interpret such findings as evidence for a kind of peristaltic movement that takes place in a cranio-caudal direction is against all of our physiological

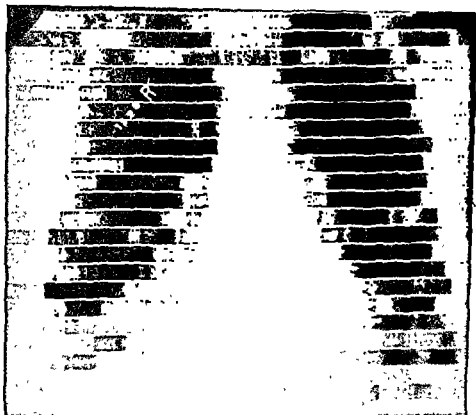


FIG 49—Graphic registration of movements of silhouette borders in a healthy individual. Simultaneous registration by means of multiple-slit kymography (method of Stumpf). Graphs appear in horizontal bars. By laying off on the waves of each bar a definite distance and connecting these homologous points, the shape and outline of the entire silhouette at any particular instant may be shown. Courtesy Dr. I. Seth Hirsch, New York City.

knowledge because the excitation wave reaches all ventricular parts in less than 0.01 sec. It is true that the movement of the various points takes place with a time interval that can easily be perceived by the eye. But that merely proves that the movements of the shadow border are influenced by factors

In the apical region the waves are more roundish since the apex moves not only

in the blood pressure as the left ventricle empties its blood into the aorta. The ascending aorta will be drawn caudad and to the left when the expulsion period is finished. The maximum right-sided movement is always found to precede the maximum left-sided movement but this time interval is not constant; it is smaller during inspiration and greater during expiration. This phasic displacement between right and left contours diminishes with a marked aortic effect, as with a high pulse pressure, while a lengthening is found with a marked ventricular effect, as with a high position of the diaphragm or cardiac enlargement with increased cardiac amplitude.

When kymograms are correlated with simultaneously made electrocardiograms, one must consider an experimental error of about 0.03 of a second; this is caused by the width of the slit. In comparing the ventricular complex of the electrocardiogram with the left lower contour in the anterior view, one author has stated that the peak of the kymographic wave begins on the average 0.09 of a second after the beginning of the ventricular complex and may occur as late as 0.13 of a second after it. This relationship does not seem to be so simple since other investigators have found the following: 1) that the caudal portion of this contour may start its medial movement with the beginning, and end it with the end of the ventricular complex, during which time a lateral movement was noted in the more cranial portions. At the same time the right lower contour moves opposite to the left caudal portions (pendulum movement); 2) with the same or a similar type of movement of the left lower contour the right lower contour may move laterally only in parts; 3) the caudal parts of the left lower contour move laterally while the cranial portions move medially. The end of the T wave of the electrocardiogram corresponds in time to the trough of the graph, which represents the change from the emptying to the filling phase of the heart.

The heart sounds coincide in the following manner with the kymographic tracings. The first heart sound occurs within 0.05 sec. of the peak of the ventricular wave. The second heart sound corresponds to the deepest portion of the trough of the ventricular graphs (aorta, pulmonary artery).

**DENSITOMETRY.** It can be demonstrated that the densest area in a heart film is quite a distance to the left of the midline. Comparison of the ionographic curve, as obtained with the previously mentioned ionization chamber, with the heart border simultaneously recorded by means of a roentgen cinematogram and electrocardiograms, shows that the change from the minimum to

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are displaced along the film in the same way that the slots are displaced in making the film. This can be done in one of three ways. First, by carefully reading from a time tracing on the film the time displacement from an arbitrary zero point of each one of the cardiovascular border graphs and then making the proper time shift as indicated by the time displacement of each individual slot. Second, by making use of a mask to align the

cycle. Or third, by cutting the film in longitudinal strips corresponding to the bars and shifting these strips so that the black reference marks are arranged transversely in one line. Then all points on any transverse line will be in phase.

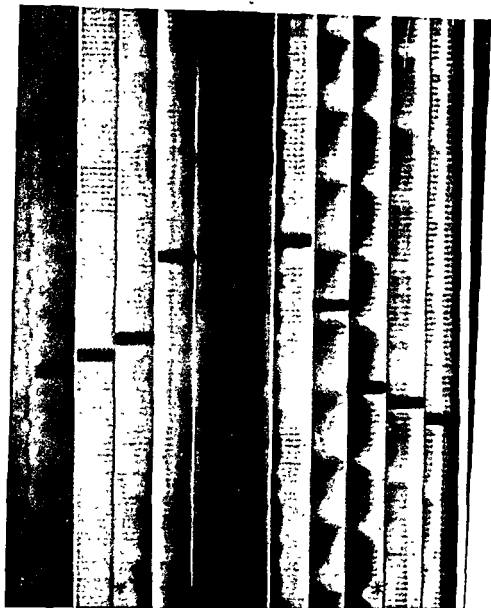


FIG. 50—Graphic registration of movements of silhouette borders in a healthy individual. Simultaneous registration by means of multiple, interrupted slit kymography. The black horizontal bars indicate level of different slits. Graphs appear in corresponding vertical bars. Respective contours from rt to lt, starting and ending with \* rt lower cardiac.

the film. Such points have not occurred at the same instant of the heart cycle because the slots do not lie on such a line. To compare positions of the cardiovascular border at any one instant of the heart cycle, it is necessary to measure the shadow at points which

in the blood pressure as the left ventricle empties its blood into the aorta. The ascending aorta will be drawn caudad and to the left when the expulsion period is finished. The maximum right-sided movement is always found to precede the maximum left-sided movement but this time interval is not constant; it is smaller during inspiration and greater during expiration. This phasic displacement between right and left contours diminishes with a marked aortic effect, as with a high pulse pressure, while a lengthening is found with a marked ventricular effect, as with a high position of the diaphragm or cardiac enlargement with increased cardiac amplitude.

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the maximum of heart filling is nicely correlated with the T wave in the electrocardiogram.

**STROKE VOLUME.** The problem involved may be stated as follows: does the visible volumetric diminution of the heart closely correlate with the total stroke volume or a constant fraction thereof? The answer has to come from roentgenologic studies carried out parallel with gasanalytic determinations. A few critical remarks and qualifying statements are appropriate.

The roentgenologic method of determining the volume of the heart quite frequently starts out from the frontal area of the heart. In an attempt to determine the true area one must take into consideration several points. First, a correction must be obtained because of the finite target-film distance; when using the kymographic method, the distance is perhaps 165 cm. (66 inches). Second, a correction must be obtained for the object-film distance; this requires measurement in a lateral view. Third, the cranial and caudal silhouette borders can be determined only arbitrarily. There should be added the fact that the ratio between the frontal area and the depth of the heart, as determined for different individuals, is not constant (see chapter IV).

It has been stated previously that the movements of the shadow borders are influenced by factors other than volumetric changes, namely, changes in shape and position; and that the study of the lateral movement of the heart, as registered by kymographic procedures, does not take into consideration that silhouette points move obliquely to the plane of observation and obliquely to the slot, i. e., not at right angles to the direction of motion of the film. Furthermore, changes in shape and size in the sagittal (antero-posterior) plane. It has also been pointed out previously that the marginal movements of the cardiac silhouette express only a part of the total volumetric changes, the other and more important part being represented by the longitudinal shortening of the ventricles. . . . . 20% of the  
ollic vol-  
l include

the right atrial shadow, would be greater than would be calculated from the ventricles alone. It should be remembered in this connection that the atria are displaced by internal stress. The difficulty of estimating the systolic diminution of the ventricular lumina from the degree of marginal movements is exemplified in the following observation. during fluoroscopic examination a patient with a left-sided pneumothorax showed the marginal movements at the left side becoming small during inspiration in association with a fall in the intrathoracic pressure, and there appeared wide marginal excursions during expiration when the intrathoracic pressure rose. The stroke volume was determined for a few seconds during respiratory standstill for both phases of respiration, and the figures for inspiration and expiration were practically the same, namely, 36.5 cc. and 37.5 cc. respectively. Finally, it has been realized and should be kept in mind that an aortic or mitral regurgitation leads to a leak that has been calculated to range from 14 to more than 50% of the gross cardiac stroke.

In a given kymogram the peaks indicate diastole, the valleys systole, and

the base line corresponds to the bottom of the exposed strip in which the reference notch occurs. The distance of the bottom of the valley from the base line is measured with dividers, and it represents the same moment of time in each of the exposed strips. This distance is laid off on each strip on both sides of the heart, and the points of intersection of the heart shadow with this distance represent the contour of the systolic projection. The diastolic outline is similarly obtained by using as reference the outermost peak of the same notch taken for the systolic reference. One may also correlate a kymographic curve with an electrocardiogram taken simultaneously. The beginning of the isometric contraction corresponds to Q or R of the initial deflection; the beginning of the ejection period is noted at the beginning of the sudden outward thrust in the aortic graph, and the end of the ejection period is marked by the second heart sound that has a fairly close relation to the end of the T deflection (exceptions occur). One draws in the exposed strip a vertical line through the peak of the ventricular curve that corresponds to the beginning of ejection (as determined from the aortic curve), and a vertical line from that point at the bottom that corresponds to the Q or R in the E.C.G. The difference between the foot point of these two lines represents the ventricular volumetric difference at the beginning and end of the ejection period.

An attempt to arrive at a formula for the stroke volume of the heart could take into consideration some of the difficulties pointed out above while other unknown quantities had to remain untouched. The formula that suggests the stroke volume of the heart, based upon roentgenologic data, reads as follows:  $0.63 \times A_d^{1.45} - A_s^{1.45}$  (where  $A_d$  and  $A_s$  represent the diastolic and systolic areas in the frontal plane). When applied for individuals without valve defects, an excellent correlation was obtained with the stroke output as determined by the acetylene rebreathing method.

It would seem logical to determine volumetric differences of the heart from a plane that shows the greatest amplitude of pulsations, and this is the left anterior oblique view.

**Summary.** Though fluoroscopy provides valuable information, the detailed analysis of the pulsations as well as their correlation (electrocardiogram, heart sounds) rests with the graphic methods. Of these, single-slit roentgenkymography shows the maximum of detail. In order to compare the activity of many points of the silhouette simultaneously, multiple slit or surface kymography, cinematography and to some degree instantaneous roentgenograms taken at determined phases are used. The correlation of the shape and the size of the silhouette, as taken in different views and at identical moments of the cardiac cycle, would be of great interest and value. This remains to be done.

The functional evaluation of pulsations rests also, in the main, with further investigation. Limitations exist due to the fact that first, the visible pulsations depict only a part of the total mechanical action and second, that they are influenced by the rotation of the heart as well as by forces acting on the surface of the heart.

The roentgenologic appearance of the cardiovascular pulsations is complicated, partly on account of intrinsic factors, partly on account of conditions

of projection. Under normal conditions only the outer pulsations can be studied. Those of the atrio-ventricular septum, of the medial and inner contours of the aorta, pulmonary artery and superior vena cava, of the supra-valvular portion of the ascending aorta and of a part of the lower heart border are not visualized and therefore are not available for study. Movements of a limited portion of the medial contour of the arch may be studied after visualization of the esophagus by means of barium paste. The visible pulsations express mainly volumetric, pressure and rotatory changes and are modified by different factors such as pericardial covering, position and intra-thoracic pressure. As a whole, one type of movement may be correlated with the emptying, another with the filling period of the ventricles. During the emptying period the heart silhouette is more globular and its base is more caudal. It may also show either a true or an apparent pendulum movement from the left toward the right side of the chest. Points near the interventricular septum show a smaller amplitude. The aorta shows a displacement as a whole as well as changes in diameter. Neither the medial nor the lateral movement of the whole left contour, which corresponds to the ventricular region, takes place simultaneously. The largest pulsations occur along the dorsal ventricular contour. The movement of the right heart border is quite variable by reason of the fact that atrial and ventricular actions conflict. The pulsations along the right vascular contour are either of a venous, arterial, or mixed type.

**Adjacent Organs. Esophagus.** The fluoroscopic and kymographic study of the barium-filled esophagus reveals definite displacements at the level of the ventricular mass, mainly in the dorsal and ventral direction—a kind of pendulum movement. At the level of the left atrium one notices rather variable findings in different people. Fundamentally the dorsal movement seems more rapid, it probably consists of two waves, one shortly preceding the ventricular mechanical systole which may be caused either by the atrial contraction or by transmission in connection with the ventricular period of rising tension, the second, smaller one, apparently takes place between ventricular systole and diastole and is perhaps caused by the diastolic atrial enlargement.

**Liver and Diaphragm.** The kymographic study and fluoroscopic observation of the diaphragm shows the presence of small pulsations on both sides. The medial portion of the left leaf, in the immediate vicinity of the heart, and as seen from in front, moves in the same direction as the visible portion of the heart: cranial during systole, caudad during diastole. This is best noted during forced inspiration and also with the vertical type of heart. The findings during forced expiration and with the transverse type of heart remain to be investigated. The right leaf shows a more rapid caudal movement

the jugular vein; the cranial movement corresponds to its 2nd wave. The caudal movement of the diaphragm results in a slight reduction of the thoracic volume during ventricular systole.

*Lung vessels* A chest film taken during respiratory standstill with an exposure time of perhaps  $\frac{1}{2}$  second shows considerable blurring of both the cardiovascular borders and all intrapulmonary vascular shadows. If several chest films of the same individual are made, with an exposure time of  $\frac{1}{15}$ - $\frac{1}{20}$  sec., some will show blurring, especially of the hilar vessel shadows, some will not. This blurring will be noticed in none of the various chest films made in  $\frac{1}{40}$  sec. If films with an exposure of  $\frac{1}{15}$ - $\frac{1}{20}$  sec. are now taken at determined moments of the cardiac cycle it is seen that the intrapulmonary blurring is most pronounced at the time of the systolic discharge and absent during the diastolic period. If the technical set-up permits, roentgenograms of the chest should therefore be taken at the moment in which the ventricles are almost at the end of the diastolic phase. Exposures made during systole likewise show the hilar vessels larger in size than in films made during diastole. The movements of the intrapulmonary vessels are partly a displacement by the mechanical movement of the heart (this holds true especially in the central portions of the lungs) and partly volume changes and changes in their course. They are perhaps also caused by vibration within the elastic lung tissue.

*Blood Flow.* This has been studied by introducing a contrast substance in the vessels of animals.

If a few  $\text{cm}^3$  of 10-30% bismuth oil are injected into the femoral vein of dogs, the centripetal course of the artificial embolus is stopped with each ventricular systole. In the right heart a whirling movement takes place, the ejection is not complete with one beat. This could indicate that the ventricle normally does not completely empty; however, it should be remembered that we are dealing with a substance of higher specific gravity than blood. The same argument holds true for the process of embolization of the lung where one finds that the basal portions are favored (more so on the right side) because changes in the position of the body demonstrate the influence of gravity.

Seventy per cent sodium iodide or abrodil was injected into the superior vena cava of dogs and roentgenograms were taken during both systole and diastole. The anterior view shows the right atrium to be almost vertical in shape during ventricular systole and crescent shaped during diastole, with the auricula leaping over the infundibulum of the right ventricle. The right ventricle during systole has a hornshaped appearance, at the height of systole the infundibulum and the very apical portion contain contrast-substance while in the middle portion the walls seem to be in contact. During diastole the lumen of the right ventricle is much larger, its outer border convex, its inner border vertical. In the lateral view during ventricular systole there are seen both the injected coronary arteries and the funnel that is formed by the closed atrio-ventricular orifice. During diastole a large column of contrast-substance is seen extending through this orifice. The pulmonary artery is smaller. Fluoroscopy shows cloudy shadows moving from the base toward the apex where they turn around and disappear cranially. When the animals are dying, the contrast-substance scarcely enters the right ventricle and enters not at all into the pulmonary artery, while the atrium forms a wide sac.

Thorotrast was injected into the jugular vein of dogs and cats. The amount was  $\frac{1}{2}$ -2% of the body weight, and the duration of the injection was several



minutes. Then the right ventricle was noted during systole to become smaller and to shorten in its basal portion by about one-third. Following systole blood shot from the right atrium into the right ventricle—first in a thin stream and subsequently in the form of a wide band. The wedge or tongue-like shadows of the auricular appendages enlarged during ventricular systole and apparently took up complementary spaces. The shift of the aorta between the systolic and diastolic positions was as great as the size of the lumen proper.

Fifty per cent sodium iodide was injected into the femoral and external jugular veins of dogs, cats and rabbits, and the size of superior and inferior vena cava, as influenced by respiration, was studied by roentgencinematography. An inspiratory constriction of the inferior vena cava at its exit from the diaphragm, and narrowing in its intrahepatic portion, were definitely demonstrated. In another series of experiments thorotrast was injected directly into the wall of the vena cava in dogs. This permitted the study of any changes in diameter. No significant changes in the diameter of the thoracic portion of the vena cava were observed when the dog was lying on its side, but slight changes in diameter occurred during the respiratory cycle when the dog was standing. Conclusions as to conditions in man cannot, however, be drawn from this because dogs and cats have a muscular band, and rabbits have bundles of collagen fibers which loop over the inferior vena cava at the caval foramen.

The bulbus of the aorta was injected in rabbits by introducing a catheter through the carotid artery. Roentgenograms and electrocardiograms were correlated and it was demonstrated that the coronary arteries very probably filled mainly during systole.

Venous air embolism permits the visualization of a collection of air in the cranial portion of the right ventricle with a bulging out of the adjacent wall.

In man, the course of a contrast-substance in the veins has been studied. The veins of the arm show definite changes in their lumen for which respiration, muscular movements and perhaps chemical stimulation are responsible. The blood does not necessarily flow along the shortest possible route toward the heart. It may deviate near the narrowed portions into smaller branches, thus pursuing a zig-zag course. The flow in the axillary veins is accelerated. Contrast-substance was injected into the saphenous vein and the emptying time for the recumbent position determined, it was 5 to 30 seconds in normals, but up to 2 minutes when there had been bed rest for a good number of days. This slowing was counteracted by inhalation of  $\text{CO}_2$  and was not observed at all in patients affected by thyrotoxicosis. People with well-developed musculature of good tonus have a rather rapid venous current. Elevation of the leg, and muscular contractions will always increase the velocity of the current.

As to the intracardiac injection of contrast-substance in man, see chapter I.

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## CHAPTER V DYNAMICS OF THE NORMAL CARDIOVASCULAR SYSTEM

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## CHAPTER VI

### ANATOMICAL—CLINICAL—ROENTGENOLOGIC CONSIDERATIONS

**Palpation, Percussion and Auscultation.** The principles upon which palpation and percussion depend differ from those upon which the roentgen silhouette is based. It is therefore erroneous to expect identical results when applying these two methods. The roentgenologic projection is independent of conditions between the silhouette of the heart and the place of projection. It is an objective method. The status of the heart action, the distance of the heart from the surface, as well as individual differences in lung volume, curvature and elasticity of the chest, etc., influence the results of the subjective methods of palpation and percussion. The clinician who, with full knowledge of the differences and limitations, checks his percussion with the results offered by the roentgenologic method, will attain a much greater degree of accuracy. Perhaps it may be said that before orthodiagraphy was introduced there was no uniformity of opinion among clinicians as to the shape or extent of the cardiovascular dullness.

When a radiopaque landmark is fixed over the apical thrust, one notices that for perpendicular roentgen projection it seldom coincides, with the left lower pole region, although it tends to coincide with the left heart border. If the apical thrust is defined as the definite forward lift, imparted to the chest wall at a right angle, it is usually found to be located 1 to 4 cm. above the roentgenologic lower pole and it is approximately thrice as frequent outside the silhouette as inside. It follows then that there is clinically a greater tendency toward overestimation than toward underestimation of the heart size. This projection of the apical thrust beyond the silhouette is especially marked in two conditions. First, in the presence of a narrow chest where the lateral chest walls slope backward; second, when the transmission of the impulse radiates due to overactivity of the heart (thyrotoxicosis, exercise).

In order to check the results obtained by percussion one may proceed to fasten short pieces of wire along the outline of the cardiac dullness and make an orthodiagram or a teleroentgenogram preferably at 300 cm. distance. A simpler method is to mark the outline of percussion by means of a colored pencil and to draw with a pen orthodiagraphically the outline of the silhouette directly upon the skin of the individual. Caution is required when the ordinary orthodiagraphic tracing sheet is compared with the percussion outline on the chest of the patient. It leads to error if the sheet is placed in direct contact with the chest, especially if the tracing of the clavicles is directly superimposed upon the clavicles per se. The transparent sheet must be held vertically and must touch the chest only in the lower sternal region. The correct height is obtained if by process of visualization, the clavicles on the orthodiagram are brought into the same horizontal level with the clavicles themselves. In correlating the clenched fist with the orthodiagraphic sil-

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emptying period of ventricular systole; second, if the senses of touch and vision are acted upon simultaneously by a stimulus, it will require approximately 0.01 sec. longer for it to be perceived by the eye. This law holds for optimal visual stimuli, while with the poor intensities upon the screen the delay is greater, for the latent period between the advent of the stimulus and the beginning of the sensation increases with a decrease of the stimulating light. If now, while observing the fluoroscopic activity of the heart, one listens simultaneously to the heart sounds, an even more puzzling fact is observed. The first sound is heard during (roentgenologic) diastole, the second sound during (roentgenologic) systole. The explanation is fundamentally similar to the previous one, the first sound occurs while roentgenologically the silhouette is still large, i.e., before the emptying period begins. At the end of this period, the second sound is produced, while the silhouette still remains small since the ventricles are not yet relaxed sufficiently to be filled from the atria. Second, if stimuli reach eye and ear simultaneously, the perception by the eye is delayed approximately 0.04 to 0.06 seconds; and again the delay is actually greater because we are not dealing with optimal but rather with poor light intensities. Another psychological fact is here to be considered: with the stimulus levels for touch and light and for sound and light equal, we will perceive first that sensation to which we attend.

**Physical activity.** It can be demonstrated in animal physiology from the heart weight

ratio that the mass of the heart is directly correlated with the body weight

functional demands. Related to the same body size, birds have hearts nearly twice as large as mammals. The highest ratios in birds are found in those species which are distinguished for powers of flight or running or for loudness of singing. Comparing birds of the same species, one finds that the athletic members have much higher ratios; thus the ratio for the tame duck is 0.6-0.7% and for the wild duck 1.06-1.1%. If one excludes, among the mammals, the smallest representatives, all of which have high ratios because of the high metabolic rate, one can divide the remainder into two classes: those which are capable of continuous and severe muscular exercise and those which are not. The ratio in the former group is above 0.6% and in the latter below 0.6%. Bats have exceptionally heavy hearts; their hearts are as large as those of birds of similar size and the ratio of 1.21% surpasses that of the deer (1.15). Athletic members again have higher ratios. The average ratio for racing greyhounds was found to be 1.34% (with a maximum of 1.73%) while the average ratio for mongrel dogs is 0.8% (with a maximum of 1.0%). The average ratio for thoroughbred race horses was found to be 0.92% (with a maximum of 1.1%), while the average ratio for ordinary horses is 0.68%. Selection seems to play a rôle because greyhound pups and yearling race horses have a ratio already above the average. The ratio for the hare is three times that of the rabbit. Animals with powerful hearts have relatively extensively developed right ventricles.

In order to evaluate the effect of exercise on the heart, animals have been submitted to certain exercises for a period of 3-6 months in 24 hours. The heart v



houette, the sheet is turned counter-clockwise, until the oblique diameter is identical with a diameter of the fist, which extends from its ulnar and proximal end to the interphalangeal joint of the index finger.

Approximately identical results may be expected, theoretically, for both percussion and Roentgen projection. This is fulfilled for an area extending several cm outside each parasternal line. The normal vascular mass, however, is too far distant from the anterior chest wall to produce, under normal conditions, any appreciable dullness. The right heart border is likewise rather deep, the wall of the right atrium very thin, and considerable lung tissue overlies it. Therefore on percussion of the right heart its border is located apparently more medial than the roentgenologic border. At the left side, in the third intercostal space and over the fourth rib, corresponding to the waist of the heart and to the upper portion of the ventricular region, there is usually excellent coincidence. Farther out and downward, the direction of percussion and of the roentgenologic projection diverges more and more and consequently the difference in the results obtained by the two methods becomes marked. It is a common error to place, by percussion, the left contour too far to the left and too far cephalad. This applies to both normal and pathological hearts. Underestimation, however, is the rule in the presence of emphysema.

A number of authorities have determined the average and maximal differences between the results of the two methods. Individual methods of, and capacity for, percussion as well as the use of the two roentgenological methods (orthodiagraphy or teleroentgenography at about 200 cm. distance) explain some divergence in results. On an average, one may state that in 50-60% there is agreement within a limit of 0.5-0.75 cm. Experienced examiners have found maximal deviations of 2-2.5 cm. The average difference for each side is roughly 1-1.5 cm. but it is usually greater in enlarged hearts, in older people, in women, in the presence of emphysema, obesity and deformity of the chest. Wherever the roentgenological method is available diagnosis by the method of percussion does not hold its former significance.

It can be mentioned only briefly here that clinical methods may give information not obtainable roentgenologically. There follow two examples. In the presence of right ventricular hypertrophy, and for the anterior view there may be almost normal figures in, and but moderate change in the shape of, the cardiac silhouette; but the palpating hand feels the pathognomonic sign of a heaving lower sternal portion, provided that there is an associated enlargement of the left atrium. The left lower pole region of the silhouette of a high grade mitral valvular lesion, may reveal roentgenologically no visible pulsations and yet movement can be seen or palpated on the chest wall.

If one observes the fluoroscopic activity of the heart and palpates the apical thrust simultaneously, an observation is made which is perhaps somewhat unexpected: the apical thrust precedes the inward movement and with higher heart rates it seems to occur while the heart is moving outward. This inward movement coincides with the radial pulse which is known to follow 1/10 sec. after the ventricular systole. The explanation for these observations is as follows: first, it should be recalled that the apical thrust corresponds to the period of rising tension in the ventricles and represents the very early part of the systole while the "roentgenologic systole" corresponds to the

emptying period of ventricular systole; second, if the senses of touch and vision are acted upon simultaneously by a stimulus, it will require approximately 0.01 sec. longer for it to be perceived by the eye. This law holds for optimal visual stimuli, while with the poor intensities upon the screen the delay is greater, for the latent period between the advent of the stimulus and the beginning of the sensation increases with a decrease of the stimulating light. If now, while observing the fluoroscopic activity of the heart, one listens simultaneously to the heart sounds, an even more puzzling fact is observed. The first sound is heard during (roentgenologic) diastole, the second sound during (roentgenologic) systole. The explanation is fundamentally similar to the previous one; the first sound occurs while roentgenologically the silhouette is still large, i.e., before the emptying period begins. At the end of this period, the second sound is produced, while the silhouette still remains small since the ventricles are not yet relaxed sufficiently to be filled from the atria. Second, if stimuli reach eye and ear simultaneously, the perception by the eye is delayed approximately 0.04 to 0.06 seconds; and again the delay is actually greater because we are not dealing with optimal but rather with poor light intensities. Another psychological fact is here to be considered: with the stimulus levels for touch and light and for sound and light equal, we will perceive first that sensation to which we attend.

**Physical activity.** It can be demonstrated in animal physiology from the heart weight

body weight ratio that the mass of the heart is directly correlated with the functional demands. Related to the same body size, birds have hearts nearly twice as large as mammals. The highest ratios in birds are found in those species which are distinguished for powers of flight or running or for loudness of singing. Comparing birds of the same species, one finds that the athletic members have much higher ratios; thus the ratio for the tame duck is 0.6-0.7% and for the wild duck 1.06-1.1%. If one excludes, among the mammals, the smallest representatives, all of which have high ratios because of the high metabolic rate, one can divide the remainder into two classes. Those which are capable of continuous and severe muscular exercise and those which are not. The ratio in the former group is above 0.6% and in the latter below 0.6%. Bats have exceptionally heavy hearts, their hearts are as large as those of birds of similar size and the ratio of 1.21% surpasses that of the deer (1.15). Athletic members again have higher ratios. The average ratio for racing greyhounds was found to be 1.34% (with a maximum of 1.73%) while the average ratio for mongrel dogs is 0.8% (with a maximum of 1.0%). The average ratio for thoroughbred race horses was found to be 0.92% (with a maximum of 1.1%), while the average ratio for ordinary horses is 0.68%. Selection seems to play a rôle because greyhound pups and yearling race horses have a ratio already above the average. The ratio for the hare is three times that of the rabbit. Animals with powerful hearts have relatively extensively developed right ventricles.

In order to evaluate the effect of exercise on the heart, animals have been submitted to certain experiments. One month old rats were kept running for a period of 3-6 months in a revolving cage, as much as 10 miles was covered in 24 hours. The heart weights of 5 animals showed a difference of 7.3-37.7%

houette, the sheet is turned counter-clockwise, until the oblique diameter is identical with a diameter of the fist, which extends from its ulnar and proximal end to the interphalangeal joint of the index finger.

Approximately identical results may be expected, theoretically, for both percussion and Roentgen projection. This is fulfilled for an area extending several cm. outside each parasternal line. The normal vascular mass, however, is too far distant from the anterior chest wall to produce, under normal conditions, any appreciable dullness. The right heart border is likewise rather deep, the wall of the right atrium very thin, and considerable lung tissue overlies it. Therefore on percussion of the right heart its border is located apparently more medial than the roentgenologic border. At the left side, in the third intercostal space and over the fourth rib, corresponding to the waist of the heart and to the upper portion of the ventricular region, there is usually excellent coincidence. Farther out and downward, the direction of percussion and of the roentgenologic projection diverges more and more and consequently the difference in the results obtained by the two methods becomes marked. It is a common error to place, by percussion, the left contour too far to the left and too far cephalad. This applies to both normal and pathological hearts. Underestimation, however, is the rule in the presence of emphysema.

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cardiac area growth curve was determined in the pre-exercise period as well as throughout the whole study for both exercised and control dogs. The ratio of cardiac volume ( $V \approx 0.44 \times A^{3/2}$ ) was determined. The results were as follows: 7 of the 8 dogs showed an increase within 3 to 5 weeks after the beginning of exercise and 5 of the 8 exercised dogs either overtook or surpassed their controls or drew away from the controls during the period of exercise. All dogs were sacrificed. As compared with the controls, heart weights increased 5% and 13% for the runners and swimmers respectively; the heart weight-body weight ratios were increased 11% and 17%; the heart weight-leg muscle weight ratio exceeded 5.4% and 14% respectively, i.e., the heart musculature had grown proportionately more than the peripheral musculature. Another experimenter kept dogs running for prolonged periods. A subsequent microscopic study revealed an increase in the size of the nuclei for both heart and skeletal muscle fibers while an increase in the size of the fibers was limited to the heart muscle.

The relatively small number of animals used in all these experiments, the type of exercise, the possible variant response of the growing and the grownup organism, and the lack of complete uniformity in the results must be borne in mind.

Rabbits which were driven to exhaustion showed no definite changes in the silhouette although postmortem studies revealed acute dilatation of the right ventricle. Another investigator reported diminution of the cardiac area as great as minus 19%. Finally, it should be added that animals which have been chased to death reveal signs of severe autointoxication but no cardiac enlargement.

The detection of possible changes in the size and shape of the heart in man under the influence of sudden or prolonged physical activity is dependent mainly on the roentgenological method.

It is of fundamental importance to realize that single or numerous feats of physical activity will elicit different responses from the cardiovascular system. A cardiac enlargement may be expected if there is cardiovascular disease present as found in the course of infections, such as tonsillitis, sore throat, grippe, low-grade rheumatic fever, or accompanying hypertensive or coronary artery disease. It is therefore advisable, after a patient has had an acute infectious disease, to warn him against undergoing any strenuous training for a period of at least several weeks, and sometimes months.

An able pathologist has recently analyzed the heart findings in 26 athletes who died during full physical activity, most of them instantaneously. He concluded that 14 instances revealed evidence of either definite or probable cardiac hypertrophy. A critical analysis reveals that 7 cases should be discarded, 6 because of definite heart disease and 1, a woman, because of a recent delivery. Out of the remaining 18, there are figures for both heart weight and body weight available in 8 instances. In some of these 8 cases, an increase in the heart weight was assumed to be present because the average figures were exceeded. Out of this series 3 cases unquestionably showed cardiac hypertrophy. In a second series by the same author, comprising 9 cases in whom death had also occurred quickly, one case showed definite cardiac hypertrophy. The ages varied between 21 and 25 years. Microscopic studies on

(average 23.3%) in favor of the exercised rats and further experiments showed that the increase in weight was related to the amount of exercise taken. Another author trained 22 rats (running in a revolving drum) for 60 days, 52-144 km. having been covered. Then all animals were retired to inactivity and small groups sacrificed at intervals. In those sacrificed immediately, the heart weight constituted 0.5-0.6% of the body weight; 8-9 days after cessation of training one found 0.42%, and the percentage had receded to normal values—0.33-0.36%—at the 48th day. A third author experimented with two groups of rats (their age is not stated); one group was wild, the other was a group of laboratory animals. They were chased around in a room daily until greatly fatigued, sacrificed in from 3 to 37 days and carefully dissected. The changes to be described were found in the laboratory rats only (9 rats with 5 serving as controls). In those animals sacrificed after the 23rd day an

increase in the total heart weight as well as in the ratio  $\frac{\text{heart weight}}{\text{body weight}}$  was observed. Elongation of the heart prevailed and the increase in mass affected the right ventricle relatively more than the left. A fourth experimenter forced 22 rats to swim strenuously. Cardiac hypertrophy invariably resulted. The right ventricle was most affected; the right atrium ranged second, and the left ventricle participated frequently but not regularly. Three rats that had been swimming, and 2 rats that had been running, were sacrificed 5 to 6 months later, and the hearts were found to have normal weights. The chief involvement of the heart was closely related to the type of exercise; swimming affected the right heart, and the effect of running was more marked in the left heart.

The  $\frac{\text{heart weight}}{\text{body weight}}$  ratio of the wild rabbit is 0.33%. After caging it was reduced to 0.25% and approaches that of the tame rabbit (0.24%); there was no corresponding loss in skeletal musculature.

The following experiments on animals are of interest. Five dogs were required to walk thrice daily on a motor-driven treadmill a distance of 2-3 km. and 1000-1400 m uphill in  $\frac{1}{2}$  to  $\frac{3}{4}$  of an hour. As compared with 3 controls, it was found that skeletal and cardiac muscle hypertrophied to the same extent, i.e., the ratio remained unchanged. Other experimenters, however, obtained different results. Five dogs, 6 to 18½ months of age, were kept running on a treadmill for a total of 120-414 hours over a period of 3-6 months. The ratios—average 0.84%—were definitely greater than those of their unexercised litter mates—0.56%. The changes were greater in the growing dogs as compared with the adult dogs. Another investigator used 6 young dogs of the same litter, four as controls, one in a cage, while the sixth was allowed to play all day long, and for the last two months had to walk for hours up to elevations of 200 m. The lateral orthodiagraphic areas for the resting and exercised dogs were 30 and 35.5 cm<sup>2</sup>. respectively and the ratios 0.55% and 0.62% respectively. Roentgenographic observations were reported on growing dogs, some of which were caused to exercise as long as 1½ years, running on a tread wheel and swimming with weights loaded about the shoulders, while litter mates were held as unexercised controls. The

should be borne in mind that if a group of athletes are examined roentgenologically, immediately following the end of exercise, a different time will elapse for each of them until his record is taken. Where exercise is exhaustive, both the systolic and diastolic blood pressure may fall considerably below average normal figures. 5) it is almost impossible to make a roentgenological study while the exercise is continuing. Exceptions, however, are certain exercises such as elevating a leg, swinging dumb-bells, or muscular activity while riding a bicycle ergometer. Orthodiagraphy and teleroentgenography at determined phases and cinematography have shown no or only very slight plus or minus changes. In the latter cases errors in technique may account for this. Kymographic studies have shown an increase in the amplitude of pulsations during exercise of 40-60%. The heart rate during exercise rarely increases more than three times the original. This, in connection with the limited increase of the visible amplitude, can hardly be assumed to explain the enormous increase in the stroke volume unless the movements of the atrioventricular septum increase very considerably. The fact that the normal heart does not increase in size during exercise, is incompatible with the prevailing view of the physiologists, namely, that the increased afflux of venous blood during diastole produces a temporary dilatation of the ventricles of the heart, so that the muscle fibers are lengthened prior to the systole and consequently enabled to contract more powerfully. Rather it would seem that the greater amount of work is accomplished by an increase in the emptying power and a decrease in the rest volume of the heart. Nothing is known as to the size and pulsations of the heart when the dead point is reached and during the phase of the second wind.

The clinical concept of an acute, almost physiological cardiac enlargement, following very strenuous exercise, was disproved 20 years ago by roentgen study. The diffuse apical impulse, changes in the position of the ribs and of the diaphragm, and in the air-filling of the lungs had misled the clinician. Immediately following completion of athletic exercise there is a moderate diminution of the cardiac silhouette. This was observed in such a high percentage and in so many instances that it cannot be accounted for on the ground of erroneous interpretation due to faulty technique. This diminution is expressed in T and L and by volumetric determination by means of the formula  $A \times T_D \times 0.63$ . One would be inclined to attribute it to an increased discharge. But since this diminution was found to persist, even for a great number of hours following very strenuous exercise, other factors (tonus?) must play a rôle. An average of minus 6-7% for linear or area measurements is given by most authorities. An increase in the size of the silhouette has been noted in individuals who display a tendency to an orthostatic diminution of the silhouette in the upright position. The deepened respiration and the increased tone of the peripheral vessels and skeletal musculature last beyond the effort period. This brings about an emptying of the peripheral blood depots, and hence an increased influx of blood results. A widening of a previously normal silhouette has been observed in persons who collapsed following effort. To this it is interesting with some myoc  
crease. Further studies should be made with material of this sort

the status of the heart musculature were not reported for most of these cases, but macroscopically there was no evidence for disease. Another case has reported, age 31, where the microscopic study revealed fibrous scars in one of the papillary muscles but nothing else. Thus there are now available 4 hearts of athletes (two of mountain climbers and skiers, one of an all-round man with preference for running, one of a physical educator who excelled in gymnastics, and one of a professional boxer) in whom the average total heart weight was 452.4 grams, and the average heart muscle weight 387 grams. These figures definitely exceed the average weight for normal hearts. The common anatomical shape of these hearts was elongation with but little or no widening.

It is interesting to recall a statistical study which showed that out of a total of 38,269 American college men, the life expectation of the 4,976 athletes compared well with that of their classmates. Constitutional inadequacies, extreme youth, severe emotional influences (shell-shock), and lack of proper physical training are either contributing or even, occasionally, etiologic factors which, in connection with strenuous physical activity, may bring about cardiac enlargement. The proof lies in the fact that therapeutic reduction, by means of rest and digitalis, is possible.

Changes in the heart size may occur during exercise, following exercise, or may be occasioned by a period of prolonged training.

For the general roentgenologic problems, the reader is referred to chapter IV, and more especially to the "criteria of comparison." Some special points are as follows: 1) the position of the diaphragm following strenuous exercise is rarely the same as before; in some instances it is lower because of increased lung volume, while in others it is higher, especially when exercise has led to complete exhaustion. The lower chest aperture is often elevated and the depth of the chest increased; this in turn may influence the diaphragmatic position. The type of respiration is likely to be more costal than diaphragmatic and, especially with exercises of speed and locomotion, the inspiratory stage is sustained. 2) the phase of the cardiac cycle is important because of the changes in the venous return and in the systolic discharge. It seems, that with marked tachycardia, there is a tendency to sketch the denser, systolic silhouette orthodiagraphically. 3) the difficulty in obtaining records during or immediately after exercise which may be favorably compared with those taken at rest is obvious. In addition the changes which are expected amount to only a few percent for bi-  
 measurements do not account for  
 the heart undergoes soon  
 has the largest volume.  
 in part for reasons which have already been discussed in chapter IV, in part

conclusions from the results which were obtained immediately following exercise as to the changes which would take place during exercise. But immediately on cessation of exercise, the venous inflow to the heart diminishes because of the cessation of the pumping action of the skeletal muscles, while the pulse rate falls more slowly. Consequently, the output of the heart exceeds the venous inflow and the diastolic volume either falls to normal or below. It also

demands the greatest  $O_2$  consumption per kg. Skiing is usually performed at higher altitudes.

In the former group (tests of speed), the roentgenological heart size is, for practical purposes, always found to be normal. In the latter group (tests of endurance), a number of healthy athletes show a tendency to relatively larger silhouettes; nearly always, however, still within the norm, if only sufficient correlation factors are considered.

Thus marathon runners, bicycle riders, oarsmen, skiers, and to some degree swimmers and ricksha pullers are the groups where the average heart size is relatively high and where single cases definitely reach the upper limits of normal. Roentgenological studies on hard manual workers gave no evidence of cardiac enlargement. Many of the hearts in athletes show more rounding of the right lower and the upper portion of the left lower contours than is customarily seen in people without adequate physical training. In skiers, often the left lower portion of the heart shadow seems rather prominent, while in swimmers some straightening of the waist of the heart may be noted. It is perhaps a post hoc propter hoc proposition to assume that athletics of this sort cause such development of the heart. It is possible that men who have hearts slightly larger than the average have a natural superiority for such forms of athletics.

It is not uncommon to observe during the period of severe training that the heart shadow increases moderately during the course of 4-6 weeks and recedes at about the same rate following cessation of training. Though it is true that slowing of the heart rate to about 60 per minute is often observed, this does not seem to explain the increase in size entirely. This increase is perhaps related to a higher vagal tonus.

It is interesting in this connection to compare certain findings in the athletic hare and the non-athletic rabbit. The former has a resting pulse rate of 60-70, the latter of 205. After vagal section the respective rates are 264 and 321, i. e., simple ablation of the vagal control enables the hare to increase its circulation 4 fold, whereas the rabbit by this means can produce an increase of only 1.6 fold.

Whether the process underlying the increase in heart size during the training period is only a stretching of the muscle fiber or whether there is also an actual growth of heart musculature, we do not know. As previously cited,

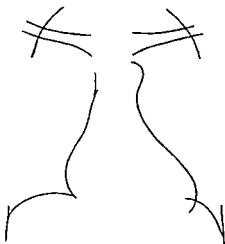


FIG 52—36 yrs, M. Perfectly well. Competitive athletics for 14 years. skating, bi-

T 14.7 cm, Th 23.3 cm



The statements as to the findings of heart size after prolonged physical activity (military service, heavy manual labor, athletics) are greatly at variance. Those who claim enlargements seem, as a rule, to have come to this conclusion either because hearts which were not normal to begin with were included, or because they compared their figures with certain standards with-



FIG 51—21 yrs, M History not significant No complaints 7 yrs of football, baseball, track and wrestling Height 173 cm, weight 79 kg., chest circumference 96 cm Apical thrust barely palpable Heart rate 56-60 B.P. 128/80 Orthodiagraphic projection of cardiac silhouette on ant. chest wall. This procedure is not as correct as an ordinary orthodiagram. The following figures refer to this orthodiagram, those in brackets to the measurement taken from the drawing on the skin. L 15.0 [14.6], B 11.1 [11.4], T 14.8 [15.2], Th 26.7 cm The silhouette just exceeds the size of the fist Heart size at the upper limit of normal, perhaps slightly exceeding it

out considering the total physical development. It should be a strict rule that any suspected abnormal findings of the heart size shall be evaluated only in connection with the total body development of the bearer. Often, too, average figures only were considered instead of the range of variation. And finally it is conceivable that a heart which was originally undersized will, under the influences of training, develop to normal size. Athletes seldom if ever show what one would call a small heart. But otherwise marked variations are observed as one might expect.

Different physiological responses are to be expected with different forms of physical activity. Tests of strength are often performed as static exercise, here belong stages in the act of weight-lifting The pumping action of continuously contracting and relaxing muscles is absent and this interferes with the venous return; in addition, the intrathoracic pressure is considerably increased. The degree of increase of intrathoracic pressure which is needed to cause a brief, marked interference with the heart size, varies apparently for different individuals Dynamic exercises can be subdivided into tests of speed, where efficiency in locomotion

is needed in a given unit of time; and into tests of endurance, where less work is accomplished in the unit of time, and a demand for efficiency in effort is made upon the muscle unit. A 100 m. race induces rapid changes in the physiology of the body but is followed by rapid regression; it can almost be accomplished by taking one single breath. Other tests of speed are represented by tennis, boxing, fencing Tests of endurance are skiing, rowing, cycling, swimming, and running for long distances. A great capacity of the heart for a prolonged increased stroke volume is demanded. Swimming, for instance,

only a part of the general problem in evaluating the functional state and capacity of the cardiovascular system.

**Pregnancy.** As one may expect, the heart shows a physiological reaction in the sense of progressive changes during pregnancy, and regressive ones following delivery. During pregnancy the basal metabolism is increased 10-20% (average); a mean of plus 10% is found for the stroke volume. The weight of the heart muscle increases without, however, surpassing normal limits, and the ratio of heart weight to body weight remains constant. The displacement of the apical thrust and the increase in dullness to percussion, which is caused by a closer relation of the mass of the heart to the anterior chest wall, easily misleads one to clinically diagnose a dilatation, especially when associated with the common finding of murmurs and accentuation of the second pulmonic sound.

The roentgenological evaluation has mainly two factors to consider. The first is the change in diaphragmatic position; the cranial shift varies from zero to 4 cm. for different individuals, with an average of 1.5 to 2 cm. The effect of this shift depends, however, upon the spatial conditions in the chest. If, for instance, the distance of the aortic arch to the upper chest aperture is short to begin with, the vessel shadows will be larger because of a wider spread; whereas slight changes occur with greater original distance. The displacement by the elevation of the diaphragm is less than one is inclined to believe since the lateral, visible portions move more cranially than the centrum tendineum does. The second factor is a change in projection. Because of the shift in the center of gravity the body of the pregnant woman assumes an increased lumbar lordosis. This causes a change in the antero-posterior angulation of the heart. Roentgenological statements of definite cardiac enlargement are erroneous and may be explained in a twofold manner. First, roentgenograms which were taken at 150 cm. were compared with standard figures obtained by orthodiagraphy; second, the pleuropericardial triangular shadow was not differentiated from the cardiac shadow and was thus included in the measurement.

Increased pulsations are commonly noted along the silhouette, including the contour of the superior vena cava. Clinically one observes an exaggeration of the jugular pulse. All these findings should be explained by the increase in the amount of circulating blood.

In taking as standard figures which were obtained at the time of three month pregnancies, it was found that until the end of the seventh month, one-third of the heart shadows showed an increase in diameters of more than 0.7 cm. There were more cases without changes than with changes. During the eighth and ninth month, one-half the total cases showed an increase. These figures were greater in the presence of functional cardiac disturbances. The increase affects T and L and the average increase of T amounts to 1 to 2 cm. Studies undertaken 8 days before and after delivery showed a decrease for the latter period of 0.77 cm. for T, and of 0.97 cm. for L. It was attempted to determine the volumetric changes in applying the formula  $V = A \times T_D \times 0.63$  and an average increase of 50 to 75 cm<sup>3</sup>. during pregnancy was found;

but the ratio  $\frac{\text{heart volume}}{\text{body weight}}$  remained practically unchanged. As compared

actual heart weight changes have been observed in animals. We also do not know whether such a possible increase in the mass of the heart musculature in man is in proportion to the rest of the musculature of the body or not.

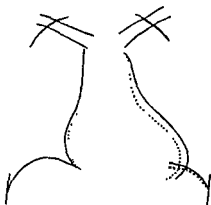
The amplitude of pulsations is relatively large but proportionate to the slower heart rate, especially during the period of training.

Many factors must be considered for their effect on heart size and amplitude. A few of these are: blood pressure, venous return, length of the diastolic period, amount of resting blood in the heart, amount of circulating blood, intrathoracic pressure, fluid loss, pH concentration, sympatheticotonic, humoral and nervous regulation.

In those cases where the size of the heart reaches the borderline, one single roentgenologic study is of limited value; follow up studies at intervals of 4-8

FIG 53—22 yrs, M History: Tonsillitis six mos. ago followed by tonsillectomy. There were eight years of competitive athletics comprising football, baseball and hockey. There were no complaints. Findings: The ht. was 167.5 cm. (5 ft 7 in.), the wt. 87.3 kg (192 lb) and the chest circumference in expiration 98.7 cm (39½ in). The apical thrust was just palpable 10.5 cm to the lt. of the midline. There was a short systolic apical murmur that was not transmitted, and  $P_2$  was not accentuated, the rate was 72/min, and the B P 138/82.

*Orthodiagrams.* The solid line tracing was taken during the training period, and the dotted line tracing after athletic activity had been discontinued for a period of four weeks. The measurements at these two occasions were as follows.



	First	4 wks later
Oblique diameter	15.7 cm	14.1 cm.
Broad diameter	10.4 cm	9.7 cm
Product of these two	163.3 cm <sup>2</sup>	136.8 cm <sup>2</sup>
Width of chest	23.6 cm.	23.9 cm

*Comment.* A regression of heart size following cessation of strenuous athletic training is demonstrated.

From Roesler, H, *Am. Journ Roentg*, 1936, 36, 349. Courtesy Charles C Thomas, publisher, Springfield, Ill

weeks will give more valuable information. Constancy of the heart size permits a liberal attitude while the tendency to progression requires strict limitation or complete cessation of physical activity. Strictest adherence to the requirements for comparative roentgenograms, as outlined in chapter IV, is imperative.

That cardiac enlargement has been and is being diagnosed roentgenologically where there was and is none, cannot be doubted. Minor deviations from the norm may escape diagnosis. In case of definite cardiac enlargement, one should consider athletic activity only as a contributing factor and search for other, primary etiology. Cardiac enlargement, important as its recognition is, is

changing the venous inflow into the heart, the blood pressure and the nervous tonus, pitressin exerts a direct effect on the coronary arteries in the form of spasm. This coronary spasm, as observed in dogs, is associated with acute cardiac dilatation. Coronary spasm, together with the fall in blood pressure, leads to a marked increase in the cardiac rate which tends to diminish the heart size; this change in cardiac rate may be counteracted by vagal paralysis (atropin). Within 2 min following a pitressin injection, there results enlargement of the heart which seems to affect the right heart more as indicated by the shape of the silhouette in the different views. T increases about 30%. The lung fields, during the acute phase of dilatation, show no signs of congestion whatsoever. The rabbit's heart likewise reacts with enlargement and simultaneous conduction disturbances

*Digitalis.* (a) Animals. 1) the cardiac area in a dog was shown to decrease from 55 to 33 cm<sup>2</sup>. under the influence of a lethal dose. This corresponds to the maximal systolic ventricular contraction. An increase near the time of the exitus is accounted for by an enlargement of the right atrium 2) dogs with cardiac enlargement due to long-standing, artificially produced mitral regurgitation, but without failure, were given digitalis. A decrease of the cardiac area and an increase of the ventricular contractions resulted. The previous findings recurred as the digitalis effect disappeared. The determination of the extent of the cardiac amplitude is made on kymographic tracings, drawing tangents to the troughs and crests and finding the average distance in a single respiratory cycle. While the conclusions drawn are independent of the magnification resulting from the central projection, the absolute figures appear increased and the magnification changes with alterations in the heart size. 3) following digitalis medication, for a target film distance of 85 cm., and with a heart rate of not more than 30 per minute, the increase in the amplitude at the left contour for 6 normal dogs was found to be 39-113%, and for the right contour 26-67%. 4) Strophantin was shown to produce in rabbits a transient decrease of the cardiac area. This was particularly marked when the heart muscle had previously been damaged by repeated injections of spartein sulphate and epinephrin chloride 5) if the heart of normal dogs is subjected to auricular fibrillation, dilatation of the heart may occur. For teleroentgenograms, it amounts to plus 9-13% for the cardiac area after one hour of fibrillation. Now if 25-50% of the lethal dose of digitalis is given (and while fibrillation still persists) the size, one hour later, will be plus 0-6% of the original value for the area. One hour later (the normal rhythm being reached) the area varies from minus 9% to plus 4% as compared with the figures before the beginning of the experiment

b) Man. The evaluation of the digitalis effect is simpler for the normal heart. Following a sufficient amount of the drug, as shown by the characteristic changes in the electrocardiogram, the cardiac area was found to diminish 7-15%. Further, an insignificant decrease in the heart rate is associated with a decrease in its output. Digitalis in the presence of cardiac failure brings about changes which complicate the roentgenological interpretation, viz., slowing of the heart rate, especially in the presence of auricular fibrillation; an increase in the output; a change in the blood distribution; a decrease in the venous pressure; a diminution in the size of the liver caused by the beginning of

with these minor and physiological changes, marked dilatations, without regression, were observed to take place in the presence of mitral valvular disease, and there was steplike progression during the course of repeated pregnancies.

An anatomico-clinical entity in the sense of heart disease in association with uterine myomata does not exist. If cardiac enlargement is found together with the presence of myoma, then hypertension, coronary artery disease or anemia must be thought of.

**Study of Pharmacological Effects.** *Amylnitrite* dilates the peripheral vessels, lowers the blood pressure, increases the heart rate, decreases the vagus tonus and increases the accelerans tonus. In the great majority of both men and cats there results a temporary moderate decrease of the cardiac silhouette. This was observed by teleroentgenograms, giving due consideration to the phase of the cardiac cycle, and by combined kymography of both heart borders. With the latter method it was shown that the amplitude does not change and the actual linear decrease has a maximum of about 11%. A marked increase in the

and nitroglycer

at an increase

the type of pulsations along the contour of the superior vena cava have not been reported.

*Atropine* causes tachycardia by decrease or paralysis of the vagal tonus. There results a decrease in the cardiac silhouette which is observed by teleroentgenograms, orthodiagraphy and kymography. The degree of the decrease depends upon the degree of tachycardia. Maximal changes for the area are minus 15-20%, for the actual linear dimension minus 14%, as obtained by different investigators and methods.

*Adrenalin* increases the blood pressure, causes tachycardia and sympathetic stimulation. The second and third factors counteract the first in its influence upon the heart size. Dilatation of normal hearts is never observed. It usually takes place in man but is observed in animals; it is likely that the same factors operate for this. In about half of the cases there is a decrease; the same decrease

is observed in the amplitude, though not regularly by any means. Hearts, which were affected by valvular lesions, showed an increase in the silhouette, this interesting fact should be studied further with coöperation between clinicians and roentgenologists. A case with Addison's disease has been reported in which the heart size increased during attacks of low blood pressure. Following adrenalin injection, the heart size decreased together with an increase in the blood pressure.

*Neosynephrin.* When 3 to 10 mgm. of this sympathomimetic drug are injected, the heart rate will slow to 30 to 45 beats per minute, and this degree of bradycardia may persist for as long as 80 minutes. The cardiac area increases from 5 to 20%.

*Nitroglycerin.* When this drug is given to patients who display roentgenologic

changing the venous inflow into the heart, the blood pressure and the nervous tonus, pitressin exerts a direct effect on the coronary arteries in the form of spasm. This coronary spasm, as observed in dogs, is associated with acute cardiac dilatation. Coronary spasm, together with the fall in blood pressure, leads to a marked increase in the cardiac rate which tends to diminish the heart size; this change in cardiac rate may be counteracted by vagal paralysis (atropin). Within 2 min following a pitressin injection, there results enlargement of the heart which seems to affect the right heart more as indicated by the shape of the silhouette in the different views. T increases about 30%. The lung fields, during the acute phase of dilatation, show no signs of congestion whatsoever. The rabbit's heart likewise reacts with enlargement and simultaneous conduction disturbances.

*Digitalis.* (a) Animals. 1) the cardiac area in a dog was shown to decrease from 55 to 33 cm<sup>2</sup> under the influence of a lethal dose. This corresponds to the maximal systolic ventricular contraction. An increase near the time of the exitus is accounted for by an enlargement of the right atrium. 2) dogs with cardiac enlargement due to long-standing, artificially produced mitral regurgitation, but without failure, were given digitalis. A decrease of the cardiac area and an increase of the ventricular contractions resulted. The previous findings recurred as the digitalis effect disappeared. The determination of the extent of the cardiac amplitude is made on kymographic tracings, drawing tangents to the troughs and crests and finding the average distance in a single respiratory cycle. While the conclusions drawn are independent of the magnification resulting from the central projection, the absolute figures appear increased and the magnification changes with alterations in the heart size. 3) following digitalis medication, for a target film distance of 85 cm., and with a heart rate of not more than 30 per minute, the increase in the amplitude at the left contour for 6 normal dogs was found to be 39-113%, and for the right contour 26-67%. 4) Strophantin was shown to produce in rabbits a transient decrease of the cardiac area. This was particularly marked when the heart muscle had previously been damaged by repeated injections of spartein sulphate and epinephrin chloride. 5) if the heart of normal dogs is subjected to auricular fibrillation, dilatation of the heart may occur. For teleroentgenograms, it amounts to plus 9-13% for the cardiac area after one hour of fibrillation. Now if 25-50% of the lethal dose of digitalis is given (and while fibrillation still persists) the size, one hour later, will be plus 0-6% of the original value for the area. One hour later (the normal rhythm being reached) the area varies from minus 9% to plus 4% as compared with the figures before the beginning of the experiment.

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diuresis and consequent change in the diaphragmatic position. After due consideration and choosing cases without liver enlargement there is still a significant increase in the amplitude of pulsations of the left lower contour; and a decrease of the cardiac area is observed to take place within 24 hours after sufficient dosage (0.8 to 1.8 grams). Simple fluoroscopic observation convincingly shows this increase in amplitude. It has also been stated that the ventricular systolic contour, which appears rather flat during the stage of failure, becomes more curved when compensation is reached. Nearly all the studies have been undertaken upon individuals with a mitral valvular lesion. Should digitalis be capable of diminishing the size of the right ventricle, then a greater part of the left ventricle should consequently participate more in forming the contour because of the clockwise rotation of the heart. This would change the amplitude and it also would very likely account for changes in the area. Further studies are therefore required on persons with left ventricular failure but with relatively little or no enlargement or failure of the right ventricle. Digitalis (1.6 to 1.8 grams) in the presence of heart disease shows no uniform effects with respect to the size of the cardiac area. A diminution of the area was associated with a decrease of cardiac output in the presence of auricular fibrillation while with sinus rhythm a diminution of the silhouette was found to be associated with either a decrease or increase in cardiac output, i.e., the heart behaved either like a normal or like a failing heart. Studies on patients with severe heart failure and normal sinus rhythm have been undertaken to test out the effect of a crystalline cardiac glycoside of digitalis lanata. When 1.6 mgm. (eight cm<sup>3</sup>) of lanatoside c were injected intravenously, the transverse diameter of the cardiac silhouette was shown by roentgenkymograms to diminish 0.5 to 2.0 cm. within a period of 30 to 60 min. And within the same time an increase was noted in the systolic and diastolic volume differences, indicative of an increase in the stroke output.

Following digitalis medication, a definite decrease in the heart area was observed in soldiers who, during the war, developed cardiac dilatation following enormous physical overexertion. Hypertension, valvular defects and abnormal heart rhythms were excluded and cardiac failure was absent; their age group was 19 to 32 years. Such observations make it seem more than probable that digitalis has an effect on the tonus of the heart muscle.

**Dilatation and Hypertrophy.** It is almost needless to point out that more than one etiologic factor may be responsible for cardiac enlargement. To cite only a few examples: In four-fifths of patients with coronary thrombosis, in

combination of emphysema and hypertension is common.

Enlargement may affect the chambers of the heart unequally. In respect to the left ventricle, the following factors may be mentioned: Hypertension (of any etiology, including acute and chronic glomerulonephritis, and coarctation of the aorta); aortic regurgitation, mitral regurgitation, aortic stenosis (hypertrophy prevails); coronary artery sclerosis; aneurysm of the wall. The right ventricle is involved predominantly in the presence of mitral stenosis;

in the late stages of congestive failure from almost any cause; occasionally in the presence of systemic hypertension without failure; chronic disease of the pulmonary parenchyma, such as emphysema; obstructive disease in the lesser circulation (sclerosis, endarteritis, chronic embolization); tricuspid regurgitation; interatrial septal defect; pulmonic regurgitation or stenosis (hypertrophy prevails); B-avitaminosis. The left atrium is particularly enlarged in the presence of mitral valvular disease; and this is especially the case when the disease results from fibrosis of the wall following rheumatic myocarditis, but is also observed with failure of the left ventricle. For the right atrium, causative factors are right ventricular failure, tricuspid regurgitation or stenosis, and the interatrial septal defect. It should be stated that the degree of enlargement in the presence of valvular lesions does not always seem to be closely correlated with the apparent extra load.

The more generalized form of enlargement is noted with a number of conditions—some common and some rare, viz., combined valvular lesions, advanced stages of congestive failure of varied etiology, myocardial lesions in the course of rheumatic fever, diphtheria, grippe, typhoid fever, Fiedler's type of myocarditis, myxedema, acromegaly, and severe and prolonged anemia. In this connection it is proper to state that all myocardial lesions of toxic-infectious, endocrine or nutritive origin may, but do not necessarily, lead to cardiac enlargement. Among other causes may be mentioned arteriovenous fistula, marked bradycardia, glycogen storage disease, rhabdomyomatosis, hemochromatosis with infantilism, transposition of the great arterial vessels, common arterial trunk, origin of one or both coronary arteries from the pulmonary artery, or bilocular and trilocular heart.

Acute dilatation of the heart is undoubtedly a rare condition. Perhaps the best instance of its occurrence is that following severe toxic damage such as noted in the presence of diphtheritic infection. The right heart may dilate subsequent to pulmonary embolism. Contrary to common belief, general enlargement does not usually follow an attack of coronary thrombosis. This re-

A healthy heart may dilate suddenly. This is clearly noted in fluoroscopy during the pause following a premature ventricular beat, likewise, when the breath is held, the glottis closed, and a strong inspiratory effort is made. Strenuous exertion never causes dilatation of a healthy heart. Paroxysmal rapid heart action may be followed by dilatation, but only in connection with the development of congestive failure. Care should be taken not to confuse acute dilatation of the heart with collection of fluid in the pericardial cavity.

While the physiologist considers enlargement of the heart muscle as a means of adaptation, the clinician is inclined to correlate these findings with potential or actual myocardial insufficiency. In the presence of coronary artery disease, cardiac enlargement is a much less valuable guide than in valvular lesions. Anginal failure is compatible with a normal heart size. Patients with coronary artery disease who develop cardiac infarction often reveal no change in heart size on follow-up studies. When this is the course of the disease, it will be



found that a fairly active life is carried on much more commonly as compared with those who either had cardiac enlargement to begin with or who developed cardiac enlargement subsequent to the attack of cardiac infarction.

Congestive failure rarely occurs in the presence of normal-sized hearts. But in this connection it should be remembered that in chronic constrictive pericardial disease the heart is prevented from increasing in size and that failure of the circulation nevertheless develops sooner or later.

Patients with cardiac enlargement have, as a rule, a diminished capacity for work. The term enlargement in this connection refers mainly to the ventricular mass. The general remark holds true that the larger the heart, the worse the exercise tolerance, experience with individual cases, however, shows excep-

more frequently, and the same holds true of the occurrence of thrombosis along the atrial wall or in the coronary arteries. It may also be stated that there is no strict proportion between the degree of enlargement and the signs and symptoms of heart failure. Heart size is not more than a fair prognostic criterion, and the clinician never should omit obtaining a correct idea as to the general exercise tolerance of a patient. Furthermore, the etiology and special anatomical features of a disease process are important items in the prognostic evaluation and should always be taken into consideration. It is known that sudden death often occurs in the presence of syphilitic coronary ostium stenosis with or without associated aortic regurgitation, or in the presence of a high degree of aortic stenosis, and that in both instances this occurs regardless of heart size. The correlation of cardiac enlargement with mortality seems to be highest for patients with rheumatic valvular lesions. In one such very careful study on 1164 cases, it was found that the mortality quota for all valvular lesions together for three groups of heart sizes, as determined by orthodiagraphy, was as follows. For approximately normal-sized hearts the ratio of actual to expected deaths was 420 percent, for moderately enlarged hearts 589 per cent, and for greatly enlarged hearts 1092 per cent.

The rate and degree of development of enlargement depends first upon the severity of the disease. The more marked, for instance, the toxic damage, the greater will be the cardiac enlargement. A second factor which contributes to cardiac enlargement is physical overexertion while the disease process is still

ever, progression takes place over shorter periods of time, and it is difficult to obtain exact information as to the rate of progression. An increase, especially if rapid, gives a poor prognostic outlook. This holds particularly true in myocarditis.

To make a diagnosis of cardiac enlargement involves a serious responsibility. An erroneous diagnosis may unjustifiably withhold normal and healthy activity from a youngster, may deprive a man and his family the benefit of a life insurance policy, and may become most unnecessarily for the patient a source of anxiety and implant in him or her the seed of inferiority feelings. But even if the diagnosis of cardiac enlargement is established beyond doubt, it will gen-

erally be wise not to use this term in conversing with the patient because what is a truth to the physician may be a nightmare to the patient.

Two anatomical forms of dilatation are distinguished, the myogenic and tonogenic type. Both types may easily be combined. The former is found with diffuse damage to the ventricular musculature and is characterized by the predominance of the width over the length figures for one or both ventricles. The latter is encountered in the presence of increased resistance ahead of the respective ventricle, the ventricular musculature is not diseased. The process seems to be reversible if the increased resistance ceases to act. This type of dilatation develops in two stages and proceeds in the direction opposite to that of the blood current. First the outflow tract, i.e., the part from the apical portion to the semilunar valves will be affected; thus, a predominant elongation results. When the right ventricle is affected, its top portion, the conus, shows the most marked changes, though the anterior portion of the septum and apical portion is elongated also. A counter-clockwise rotation of the whole heart is noted when visualized from the base. When the left ventricle is affected, its anterior wall and adjacent portion of the septum are lengthened with some widening of the apical portion. A clockwise rotation of the whole heart is noted when visualized from the base. In a second stage the inflow tract, i.e., the part from the apical portion to the respective atrioventricular valves will be affected, thus a globulisation results. The posterior portions of the wall and septum for the respective ventricles are involved. As soon as the process surpasses the apical portion, the degree of the respective rotation of the heart diminishes until it disappears completely when dilatation has reached or surpassed the respective atrioventricular ostium.

As to whether or not it is possible to differentiate roentgenologically the myogenic and the second stage of tonogenic dilatation remains for further investigation. The first stage of tonogenic dilatation, as expressed by a moderate elongation of the silhouette for the left ventricle and by a prominence of the conus region for the right ventricle, is noted roentgenologically.

The following remarks refer particularly to the roentgenological aspects of enlargement of the heart.

The principal expansion of the left ventricle is dorsad but also to the left, right and ventrad. The left lower pole portion appears drawn out in the silhouette. A marked bulge of the left lower contour may be caused by a ventricular aneurysm. The silhouette is modified when the heart is primarily in median position and here a more globular shape is found.

The principal expansion of the right ventricle is to the left but also ventrad, dorsad and to the right. Since the normal rounding of the left lower border in the anterior view is caused by the left ventricle, the left border of the silhouette will become steeper the more the right ventricle participates in the formation of the ventral heart wall. Smallness of the left ventricle has a similar effect.

An increase in the distance from the midline and an increased bulging of the right lower cardiac border cannot always be taken as a sign of enlargement of the right atrium but may be due to enlargement of the right ventricle.

The left atrium enlarges into the posterior mediastinum with some prevalence towards the right side.

If a change in the silhouette is observed to take place within a few days and, provided the criteria for comparison films are fulfilled, we may be dealing either with a change in the heart size or with the development or resorption of a pericardial effusion. Acute dilatation of the heart is far more often diagnosed than actually present; it is noted occasionally, however, as in coronary thrombosis or diphtheria. When congestive heart failure supervenes the pulmonary artery with its branches, the lung fields, the superior vena cava and the right atrium, rather than the ventricular mass provide evidence of change. Results of careful percussion make it almost certain that the right ventricle may undergo changes in size of which the ordinary roentgenogram gives no evidence; obviously, these changes take place in the antero-posterior direction.

It has been stated that a marked change of the shape of the silhouette in the course of changes in the position of the diaphragm with respiration indicates a poor tonus of the myocardium. Further investigation into this problem is needed. It is true that greatly hypertrophied hearts show hardly any changes in shape under such conditions.

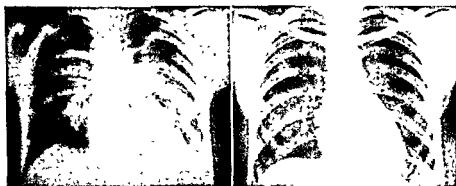
**Congestive Lung Failure.** The roentgenologic observation of the lung fields is a most valuable means of direct study of the lesser circulation. It gives information concerning the blood and fluid content of the lung tissues. It permits one to distinguish between different types and degrees of congestive lung failure and thus to evaluate the hemodynamic circulatory disproportion on either side of the lesser circulation. Positive roentgenologic findings may precede the clinical findings of rales; and signs and symptoms of failure in the lesser or greater circulation need not be concomitant.

1) One type of congestive lung failure which is comparatively rare may be characterized by: "active, hypertensive, central pulmonary, interstitial and inter-alveolar, edematous transudation; subacute" It is rather inconspicuous and deserves careful attention. It is most probably related to left ventricular failure. Azotemic cardio-renal disease (both acute and chronic) is the usual etiology. Some of the features resulting from the disturbance of metabolism in the presence of uremia are chemical irritation, fibrinous exudation and decreased tendency to resorption. These individuals have dyspnea, little or no expectoration, gallop rhythm, an increase in the systolic pressure and, unless there is aortic regurgitation present, there is increase in the diastolic pressure also. These patients are afebrile, and if crepitant rales are heard they often do not occur in basal location. Roentgenologically, the central and median-basal portions of the lungs display a cloudy opacity which is largely symmetrical but not homogeneous. It consists of wide streaks and denser, flaky and mottled areas, all hazily outlined and confluent. The transparent bands of the larger bronchi may be visible. Formations of vascular, dendritic character are not seen. The heart shadow may or may not be appreciably enlarged but its outline may be hardly visible because of lack of contrasts. The peripheral parts of the lungs remain transparent, i.e., the apical, lateral and basal-lateral areas. The opacities in the long fields may disappear quickly under medication or if, in the case of acute glomerulonephritis, anuria ceases. They may diminish if additional right ventricular failure is added to the picture.

The anatomic equivalent for the pulmonary opacities is extensive transudation of fluid into the interstices of the lungs and finally into the alveolar spaces.

Obliterative bronchiolitis has also been noted. The sites of preference are the well aerated and actively arterialized portions of the lungs. The roentgenologic picture cannot be explained by vascular dilatation, the appearance is quite different and the postmortem examination of people who die in an attack of pulmonary edema may show no overfilling of the lungs with blood. It is not a characteristic picture and, from a purely descriptive point of view, might resemble an inflammatory process or the changes that have been noted with periarteritis nodosa. The process is located centrally and it is therefore not surprising to find relatively few cases as the clinical sign in many cases.

2) Another type of congestive lung failure is not so uniform but is quite different from the first one. This might be called: "predominantly passive, hypertensive, diffuse pulmonary vascular overfilling; chronic; with perivas-



FIGS. 54, 55—18 yrs, M Moderate dyspnea and cough A few rales over the lungs B.P. 180/110 Acute glomerulonephritis with anuria Ant view moderate enlargement of silhouette, predominantly to lt. Extensive changes in the central portion of either lung

diaphragm somewhat lower also Lung fields show practically normal appearance.

cular and interstitial reaction." It varies in degree and is most typical in the presence of chronic mitral valvular lesions. The following description may be given for some of these cases. The vascular portion of the hilus is increased in size and opacity, the outlines are perhaps not distinct; the bronchial transparency is less visible. The lung fields are transparent, the vascular shadows are more prominent toward the base where there may be somewhat less contrast. A greater number of cases will reveal the following appearance: the vascular portion of the hilus shows additional vessel shadows, some in cross section, some in longitudinal section, the outlines of this whole central region are somewhat indefinite. Farther toward the periphery, the vascular shadows are not very definite and are increased in number; they may be observed in the extreme periphery. In addition, a fine areolar or marbled pattern is visible. The contrast decreases definitely toward the base and during fluoroscopy the ribs are rather poorly visualized. In other cases, multiple small nodular shadows are added to the areolar network; they may have an extensive

distribution and may perhaps be so diffuse as to leave only the apical portions free; their presence produces a fine mottled, miliary appearance of the lung fields. In other instances, hazily outlined cloudy areas of varying size and of little density are visible, and are arranged along fibrotic strands or around foci of calcification; or they may spread near the interlobar fissures, or along the periphery, or near the contours of the chest and of the diaphragm. The caudal-dorsal portions of the lungs do not increase greatly in transparency during deep inspiration. Pleural effusions may or may not be present.

The following anatomic changes account for these pictures. All the blood vessels are fuller and the blood content of the lungs is farther increased by the opening of reserve capillaries. The blood velocity is diminished more in the basal portions and in these less aerated areas the passive hyperemia, accompanied by some transudation, is most marked. Furthermore, the lymphatic vessels which are located in the interstitial tissues and around blood vessels and bronchi are congested. The hilar and intrapulmonary lymph nodes are edematous and their sinuses and adjacent lymph channels are filled with fluid. Marked transudation is found along the bronchi and larger vessels and around areas of induration and consolidation (of any etiology), along a thickened pleura and apparently wherever normal respiratory changes are interfered with. Finally, nodules which are just visible to the naked eye, represented by large groups of heart failure cells, are found in cases of mitral stenosis.

The azygos vein curves ventrad around the cranio-dorsal aspect of the right main bronchus and, in emptying into the superior vena cava, it casts a drop-like shadow that measures 0.5 by 1.5 cm. and is normally visible in about 15 to 20% of the cases. This shadow increases considerably in the course of right-sided failure but its visibility may be interfered with by hilar stasis, prominence of the superior vena cava shadow, or anatomical variations in the course of the vessel.

Pulmonary congestion that develops within a relatively short time, such as observed in the course of left ventricular failure due to hypertension, aortic valvular disease, or cardiac infarction, differs somewhat in appearance from the usual variety observed most typically with mitral stenosis. In the former, diffuse haziness, sometimes accompanied by cloudy shadows, prevails over the increase in pulmonary markings noted in the usual variety. The reason for this is edematous inhibition of the lung tissue, and here the widening of the large

appearance and  
ing fields of pa-  
tients with long-standing mitral stenosis is observed. The finely stippled appearance is also observed in miliary tuberculosis, in early stages of hematogenous metastasis, in bronchiolitis obliterans and in the diffuse type of tuberous bone formation, though in the latter case the density is very marked. A diffuse net-like appearance is also associated with carcinomatous lymphangitis and with disseminated small bronchiectasies. Mixed forms are seen with purpura and chronic purulent bronchiolitis.

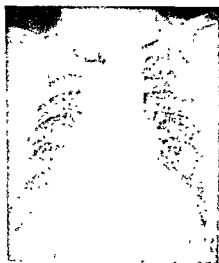
In the presence of mitral valvular disease, adequate and successful therapy will not bring about a complete regression of the aforementioned roentgenolog-

ical findings but a remarkable increase in the transparency and better definition of the vascular structures in the hilus region is noted in those instances where failure was present.

3) The characteristic indication of pulmonary edema is the presence of ill-defined, large and confluent shadows in the lung fields, with preference for the perihilar region, and this has been noted in a variety of conditions. Pneumonia is frequently and erroneously diagnosed instead. With respect to acute left heart failure we mention: cardiac infarction; hypertensive-atherosclerotic disease; coronary ostium stenosis in connection with syphilitic aortitis (with or

FIG 56—62 yrs, M History Progressive dyspnea during the past 3 mos Findings Wt 63½ kg (140 lb) Left and right sided failure was present and Cheyne-Stokes respiration was marked The apical thrust was in the 6th r sp in the ant axillary line There was a systolic apical murmur, gallop rhythm and accentuation of P<sub>2</sub>, the rate was 144/min, BP 120/80 Fundi Normal Urine with trace of albumin, urea n 31 mgm %, no anemia, Wassermann negative ECG: Sx, lt bundle branch block

*Roentgenogram Ant view* Heart enlarged and of aortic configuration Small amount of fluid at rt base Advanced degree of pulmonary congestion



*Course* The patient improved slowly on bedrest and digitalis The urea n fell to 16 mgm % The BP readings were normal at all times The wt diminished to 48.2 kg (106 lb) The Cheyne-Stokes respiration

persisted however in spite of intravenous aminophyllin medication Suddenly fever and cyanosis developed Death (3 wks following roentgenographic study)

*Postmortem* The heart weighed 450 gm The left ventricle was enlarged and hypertrophied The main coronary arteries and their central branches showed calcification and narrowing but nowhere a complete occlusion The mitral leaflets showed friable vegetations, there was no evidence of a previous rheumatic infection Microscopic The heart muscle showed an acute rheumatic myocarditis The lungs were moderately congested and there was an acute lower lobe pneumonia The kidneys showed an early arterio- and arteriolar nephrosclerosis

*Comment* The patient had atherosclerotic coronary artery disease with a superimposed acute rheumatic myocarditis, hypertension may have been present in the past. An advanced degree of pulmonary congestion is demonstrated roentgenographically

without aortic regurgitation), certain types of mitral stenosis characterized by absence of congestive failure and showing only moderate enlargement of the left atrium and persistent sinus rhythm With respect to a combined disturbance of blood pressure and salt and water metabolism, reference is made to

ditions possessing the common factor of shock (atony of the capillaries and venules, hemoconcentration) To cite examples extensive surgical intervention, burns, intoxications (from mercuric chloride, illuminating gas, rattlesnake bite, toxemias of pregnancy), internal perforations and hemorrhagic

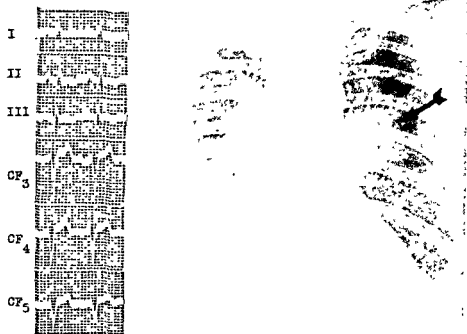


FIG. 57—68 yrs, M History 6 mos ago sudden attack of substernal oppression with radiation, lasting for one day Since then typical effort angina 6 days ago sudden onset of dyspnea followed by cough, hemoptysis, ankle edema Findings. A slight degree of cyanosis, Cheyne-Stokes respiration, slight pretibial edema and a few basal rales were noted The heart revealed gallop, apical sounds of low intensity,  $A_2$  accentuated, BP 120/95 Fundi Choroidal atherosclerosis Blood urea n 46 mgm%, creatinin 2.1 mgm%, sugar 107 mgm%, cholesterol 224 mgm%. Kidneys Urine sp gr 1032, trace of albumin, urea clearance 35% ECG NsR, normal P and P-R, initial deflection with maximal voltage in limb leads of 6 mm,  $Q_1$  present, very small initial upward stroke in  $CF_2$ ,  $S$  to  $T_2$  arched,  $T_1$  isoelectric,  $T_2$  sharply inverted,  $T$  positive in chest leads

*Ant view:* The heart is enlarged The pleura is thickened at rt Pulmonary congestion is present Adjacent to the left lung root a fairly well circumscribed area of increased density is noted (bl arrow)

*Course:* Low grade fever persisted for the first week Then the B.P. started to fall, remained below 100 and was 70/60 for the last two days of life Gallop rhythm and Cheyne-Stokes respiration persisted The peripheral edema disappeared under the influence of digitalis and mercupurin, but pulmonary rales persisted Death.

*Postmortem* (18 days following the roentgenogram) The lungs were edematous with patchy bronchopneumonia An infarct was noted in the cranial-ventral portion of the left lower lobe, pyramidal in shape and measuring 3 cm on a side The right pleural cavity was obliterated by adhesions On the ventral surface of the lt ventricle the pericardium and epicardium were adherent over an area measuring about 4 cm in diameter The entire left coronary artery was found to show considerable atherosclerosis with narrowing of the lumen The anterior descending branch of this artery was completely obstructed by an athero-  
was a myocardial infarction, F  
point of infarction, i.e., in the  
ventricles near the base, the

FIG. 58—50 yrs, M. History: Well in the past 3 wks prior to death a sudden cutting pain appeared in the rt chest with slow recovery during the subsequent week. A few days later pain of the same character in the lt chest posteriorly, aggravated by respiration, chill, no cough, no hemoptysis. Findings: Temp 39.9° C (103 4/5° F). Rapid and shallow respiration, rales at both bases, impaired percussion noted and diminished breath sounds at lt base. Normal heart sounds, rate 120/min, rhythm regular, B.P. 126/76 Wbc. 13050 with 85% polys. Blood sugar 152 mgm %, Wassermann negative. Fundi normal.



Roentgenograms, ant and lat views, illustr. omitted: Abnormal homogeneous density just above the lt leaf of the diaphragm and extending dorsad into the lt lower lobe. The interlobar septum is displaced dorsad. Fluoroscopy showed limited movement of the lt. leaf of the diaphragm.

*Course.* The temperature became normal 4 days later. W.B.C. 9050 with 77% polys.

*Roentgenogram* (6 days following the previous one): Ant view. The area of abnormal density at the lt base has diminished appreciably and partial re-aeration is noted. There remains a sharply demarcated band of homogeneous density 3 cm above the lt leaf of the diaphragm and parallel to it. Fluoroscopy showed satisfactory movement of the diaphragm.

*Course.* 3 days later the temperature rose to 38° C (100° F). Some moist rales were heard at the rt base and bronchovesicular breathing at the lt. While on the bed pan the patient developed generalized convulsions and died within a few min.

*Postmortem.* The heart weighed 550 gms, was moderately dilated and showed a marked loss of tone. The coronary arteries were about normal for this age; the valves were normal. *Microscopic.* Myocardial degeneration. A thrombus 18 cm in length was in the pulmonary artery and in its two main branches. The lt lung showed an infarct in the caudal portion of the lt lower lobe measuring 4 cm at its base and involving about 7 cm of the lateral angle. There was a diffuse reaction around it but no consolidation. There was a localized area of dense adhesions to the parietal pleura at the caudal and postero-lateral aspect of the lt lower lobe. All organs showed congestion.

*Comment.* Within a short period the patient had three attacks of pulmonary embolization of which the third one was fatal. The roentgenographic appearance of the second pulmonary infarct is fairly characteristic. If the patient had lived, a linear shadow at the lt base might have been the only remnant of the disease process.

ventral surface of the heart diagonally, involving the ventral portion of the lt. ventricular wall, and the rt side of the infarction crossing the interventricular septum, running dorsad, so that the entire apex, including its dorsal portion, was infarcted. In one area in the center of infarction so much necrosis had occurred that only the epicardium appeared to prevent rupture. The endocardium over the infarcted area was covered by a thick layer of thrombosis. Other portions of the endocardium, the valves and the right coronary artery were healthy for the age of the individual.

*Comment.* This patient was afflicted by coronary artery disease. He had a cardiac infarction in the past. The course of a very recent cardiac infarction was aggravated by pulmonary infarction. The E.C.G. findings are characteristic for the former lesion, and the roentgenogram is highly suggestive of the latter.





FIG 57—68 yrs, M History 6 mos ago sudden attack of substernal oppression with radiation, lasting for one day Since then typical effort angina 6 days ago sudden onset of dyspnea followed by cough, hemoptysis, ankle edema Findings A slight degree of cyanosis, Cheyne-Stokes respiration, slight pretibial edema and a few basal rales were noted The heart revealed gallop, apical sounds of low intensity,  $A_2$  accentuated, BP 120/95 Fundi Choroidal atherosclerosis Blood: urea n 46 mgm%, creatinin 2.1 mgm%, sugar 107 mgm%, cholesterol 224 mgm% Kidneys. Urine sp gr 1032, trace of albumin, urea clearance 35% ECG. Nsr, normal P and P-R, initial deflection with maximal voltage in limb leads of 6 mm,  $Q_1$  present, very small initial upward stroke in  $CF_1$ ,  $S$  to  $T_2$ , arched,  $T_1$  isoelectric,  $T_2$  sharply inverted,  $T$  positive in chest leads

*Ant view:* The heart is enlarged The pleura is thickened at rt Pulmonary congestion is present. Adjacent to the left lung root a fairly well circumscribed area of increased density is noted (bl arrow)

*Course:* Low grade fever persisted for the first week Then the BP started to fall, remained below 100 and was 70/60 for the last two days of life Gallop rhythm and Cheyne-Stokes respiration persisted The peripheral edema disappeared under the influence of digitalis and mercupurin, but pulmonary rales persisted Death

*Postmortem* (18 days following the roentgenogram) The lungs were edematous with patchy bronchopneumonia An infarct was noted in the cranial-ventral portion of the left lower lobe, pyramidal in shape and measuring 3 cm on a side The right pleural cavity was obliterated by adhesions On the ventral surface of the lt ventricle the pericardium and epicardium were adherent over an area measuring about 4 cm in diameter The entire left coronary artery was found to show considerable atherosclerosis with narrowing of the lumen The anterior descending branch of this artery was com-

1 cm from its origin There of the pyramid lying at the dway between the lt and rt on the lt. side to cross the

pancreatitis Pulmonary edema has been observed in patients who, during the post-operative course, were given excessive amounts of fluid parenterally.

4) Evidence of congestive lung failure need not accompany cardiovascular failure or embarrassment. This fact is observed in a good many instances in the following conditions: tricuspid valvular disease; stenosis of the pulmonary orifice or artery; constrictive adhesive pericardial and mediastino-pericardial disease; thyrotoxicosis, beriberi. A discussion of this interesting fact will be found under these respective titles.

**HYDROTHORAX IN HEART FAILURE** Hydrothorax may result from either systemic or pulmonary venous engorgement, or from the two combined. The parietal pleural veins drain into the superior vena cava or its tributaries, mainly by the azygos veins, which are also connected with tributaries of the inferior vena cava via the lumbar veins. The visceral pleura drains almost entirely into the pulmonary veins. Hence, when pulmonary drainage by the left heart is inefficient, pleural transudation will result. A relationship exists between the site of the hydrothorax and the underlying type of heart condition. A left-sided hydrothorax is favored by hypertension, left heart failure and normal rhythm. It would seem that enlargement of the left ventricle has an unfavorable effect on the circulation of the lower lobe of the left lung, either directly by compression, or indirectly by pressure on or displacement of the left pulmonary veins. In contrast to this, a right-sided hydrothorax is favored by mitral stenosis, combined right and left heart failure, and auricular fibrillation. It may be that the enlarged right atrium affects the right lung root. Pure right heart failure, such as noted with tricuspid valve disease, produces ascites without hydrothorax. The site of a hydrothorax may be further determined by local conditions such as pulmonary infarction or contralateral obliteration of the pleural space. A chronic hydrothorax often follows pulmonary infarction but may finally resolve completely.

failed to excrete sufficient diodrast from the bloodstream to cast a shadow within 70 min following the injection of the dye

*Course.* The dyspnea responded only slightly to digitalis. Within one week the urea rose to 192 mgm %, the creatinine to 14 mgm %. Convulsive seizures and uremic frost appeared. The B.P. remained high to the last. Death.

*Postmortem* (10 days following the roentgenogram) The heart weighed 550 gms. The lt. ventricle was especially hypertrophied and dilated. The coronary arteries showed no gross evidence of atherosclerosis. The valve leaflets were normal. The aorta showed a moderate amount of atherosclerosis but no gross evidence of syphilitic infection. The lt. lung was partly compressed by the large heart, and there was an infarct in the lower lobe of the rt. lung. Kidneys were pale and granular, calices and pelves normal. Microscopic: Heart: myocardial degeneration grade III, myosclerosis and chronic pericarditis. Aorta: calcifying atherosclerosis and syphilitic aortitis. Kidneys: severe benign nephrosclerosis with secondary glomerular fibrosis and atrophy.

*Comment.* An example of nephrosclerosis ending in renal failure. There was also present cardiac enlargement and failure as well as a syphilitic aortitis and osteitis. The E.C.G. revealed relatively little changes. The configuration of the heart as visualized in the roentgenogram may be designated as mitralized aortic. The lungfields reveal increased vascular injection. The hazy appearance in the rt. lower lung field is probably due to pulmonary infarction, which was found at necropsy. The roentgenographic features of pulmonary infarction are sometimes rather indistinct.



FIG 59—37 yrs, F History The patient had had 9 pregnancies and was first studied 5 yrs previously, when 7 mos pregnant again and pre-eclamptic. Findings (then). No evidence for congestive heart failure. The heart rate was 140/min, BP 235/140, Hb 13 gms, rbc 4.3 mill, urea n 35 mgm %, Wassermann negative Urine spec grav 1021, 700 mgm albumin in 100 cc, red blood cells, hyaline and granular casts Fundi. Marked angiospasm of all arterioles with few hemorrhages and exudates in the periphery. Next day expulsion of stillborn fetus Fundi. Massive cotton wool exudates and more hemorrhages but no edema of discs, a few days later absorption of exudates Course. Headache and fatigue were prominent. A few weeks prior to admission, dyspnea on effort and nocturnal cardiac asthma developed Findings Ht 152 cm (5 ft), wt 48.2 kg (106 lb) Temp 37.8° C (100° F) Congestive failure was present in both the greater and lesser circulation. The heart was enlarged, the apical thrust heaving, the lower sternum dull to percussion, and the chestwall revealed a systolic shift both to the lt and ventrad. A low grade gallop rhythm was noted, with a marked basal accentuation lt more than rt. The rate was 120/min, the rhythm regular BP 250/170 Hb 8 gms, rbc 3.5 mill, urea n 70 mgm %, Creatinine 4.8 mgm % Urine spec grav 1012, albumin 250 mgm in 100 cc, granular casts Fundi. Resolving retinitis of severe angiospastic hypertension with grade II sclerosis, a few scattered hemorrhages and exudates, a few areas of pigment proliferation, no edema of discs ECG. Sr, rate of 130 min, P-R 0.12 sec, initial deflection with increased voltage and lt axis dev, minimal depression of S-T interval in leads I, II, T of low voltage in leads I, III, of normal voltage in leads II, CF.

Ant View. Pulmonary congestion is noted, in addition there is marked haziness throughout the lower half of the rt lung field. The heart is much enlarged Roentgenograms of the skeleton showed eburnation of the rt ileum and 12th thoracic vertebra. The kidneys

Fluoroscopy is superior to roentgenography in the diagnosis of small amounts of fluid. With the former the roentgen tube may be easily centered at the level of the suspected fluid collection, and by rotating the patient, while ordering deep in- and expiration, the shifting fluid level will be readily observed. The amount of fluid at the left side is often underestimated because the diaphragm is depressed.

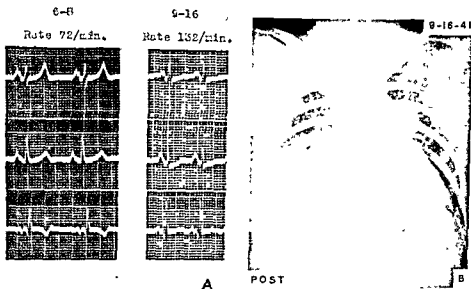
Pleural effusions are noted as a part of the disturbed water balance in acute glomerulonephritis. Their presence does not necessarily indicate heart failure.

**PULMONARY INFARCTION AND EMBOLISM.** Pulmonary infarction is an event that occurs more frequently than is commonly realized. Its clinical and roentgenologic manifestations are often erroneously interpreted. Infarction may occur in the ambulatory patient and is then sometimes diagnosed as tuberculosis, while in the bed-ridden patient pneumonia is more likely to be considered. The proper diagnosis of infarction is of practical importance. If a shadow is interpreted to be of embolic origin rather than tuberculous or pneumonic, it serves as the only warning signal of a fatal massive embolus. Pulmonary infarction in a patient afflicted by heart disease should be looked upon as a serious prognostic sign because the course of the disease will be aggravated, and not infrequently such an event marks the beginning of the end. An acute hemorrhagic lesion, in the presence of pulmonary stasis, may result from local vascular thrombosis. However, it would seem that the usual causal event is an embolus, the source of which is derived from thrombi that most frequently arise in the veins of the calf or the foot sole, occasionally in the pelvic veins and at the endocardial surface of the heart. Embolism need not cause infarction, as in the event of an acute fatal outcome. Massive embolism into either or both main branches of the pulmonary artery, if survived, may result in right heart strain due to superimposed thrombotic extension and organization. Embolism into a healthy lung may apparently merely lead to local

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and the tricuspid leaflets were thickened. The epicardium of the rt. atrium and of a part of the rt. ventricle was covered by fibrinopurulent exudate. Microscopic. The heart muscle revealed only a slight degree of fibrosis. On a longitudinal section through the rt. lung (C) one saw the lower lobe dense and deep red with a very recent and large infarction, measuring about 5 by 6 cm. in diameter. The arterial thrombus responsible for this infarction could be demonstrated. There was an old and smaller infarct in the periphery. The rt. pleural cavity contained 500 cc. of turbid fluid, and the pleura showed a fibrino-purulent exudate. There was no fluid in the lt. pleural cavity. Microscopic. No evidence for pneumonia and no growth in the bacteriological culture of pleural fluid.

*Comment.* Pulmonary infarction in a cardiac patient very often leads to a rapid downhill course. The reason for this is not fully understood. The roentgenographic appearance of the right lower lung field, in conjunction with cardiac enlargement and pulmonary congestion, was highly suggestive of pulmonary infarction, although not specific of course. As a matter of fact, the findings of high fever, high leucocyte count and pneumococci in the sputum pointed towards pneumonia. Both ECG tracings reveal the pattern of the P mitrale. The tracing taken following the pulmonary infarction shows a deep S deflection in lead I, such as noted in the course of this condition, while the appearance of the S-T intervals might have been caused by the effect of digitals and also by tachycardia.



FIGS 60, 61, 62—26 yrs, M History. Rheumatic fever at age 12 16 mos ago attack of "lobar pneumonia" with onset of dyspnea shortly afterward Findings 6 mos ago. Marked cardiac enlargement, aortic stenosis and mitral stenosis. BP 110/70, congestive failure, ECG. (6-8) N s r, rate 72/min, P deflections widened to 0.12 sec, notched, prominent in leads I, II, no axis dev, T<sub>1</sub> positive, T<sub>2</sub> negative Course Improvement on prolonged medication with digitalis and mercurial diuretics One week prior to admission the patient vomited and therefore stopped taking digitalis, and 3 days later hemoptysis started with troublesome cough followed by severe right lower chest pain Findings Rectal temp 39.5° C (103 3/5° F). Respiration 30/min in spite of morphia, cyanosis, no edema, no hepatic enlargement. Expansion of rt. chest much diminished, lower half dull to percussion posteriorly and laterally, suggestion of friction fremitus Hemoptyses Heart enlarged with precordial forward lift and broadened apical thrust in 5th I sp in ant axil line, apical presystolic and systolic and basal systolic murmur, P<sub>2</sub> acc BP 110/70 ECG (9-16): S r, rate 132/min, P<sub>1,2</sub> notched and broadened, P-R interval 0.12 sec, initial deflection with deep S<sub>1</sub>, rt axis dev (plus 125°). S-T intervals slightly depressed in leads I, II, elevated in chest leads, T deflections almost isoelectric Urine, 750 mgm albumin in 100 cc, w b c 23100 with 89% polys

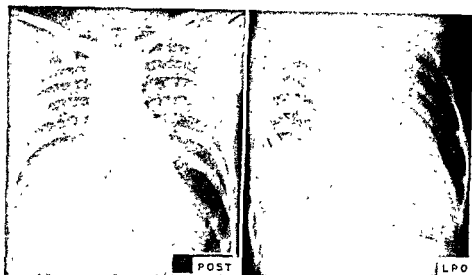
*Post. View (B)* The heart is much enlarged, revealing a combined mitral-aortic configuration Both hilar regions reveal a marked degree of congestion A large area of density is noted in the rt lower lung field It is homogeneous in character and rather sharply demarcated in its cranial and mesial aspects.

*Course:* Next day rectal temp. 40.1° C (105° F). Jaundice appeared, and the icterus index was 30 units The sputum showed type IV pneumococcus Sulphathiazol was started. One day later vascular shock and death

*Postmortem:* The heart weighed 550 grams All four cavities were enlarged and the walls thickened Stenosis of the mitral and aortic orifices was present, with calcification,

anemia, leaving the bronchial arteries to carry on nutrition. Commonly, however, and frequently in the presence of pulmonary stasis, an acute hemorrhagic lesion results. Two different sequellae are possible. The first is resolution, for which a prerequisite is a functionally normal pulmonary circulation. This is spoken of as an incomplete infarct. The second, which is the usual course of events, is organization, here the involved area shrinks and hence the size of the lung is reduced. Finally a scar results. Three quarters of all the infarcts lodge in the lower (posterior) lobes, and some preference is noted for the right side. Infarcts are often single but may occasionally be multiple. The lesions are located peripherally and thus involve one or more pleural surfaces. The sites favored are likely to be the costophrenic angles, the ventral or dorsal mediastinal margins, or the margins of the middle lobe. Since infarcts are in contact with visceral pleural surfaces, their shape is dependent upon the shape of the lung portion that is involved. The long axis of the infarct is always parallel to the longest pleural surface involved. The medial or cardiac margin of an infarct is convex towards the heart. The size of infarcts varies, depending in part upon the size of the vessel involved. The greater part of a lobe is occasionally involved, and then colliquative necrosis may set in and result in the final formation of a cavity.

From this discussion it is evident that the roentgenographic diagnosis of pulmonary infarction is rather difficult and that the interpretation of the findings will depend, in part, upon the clinical data. First of all, patients may undergo pulmonary embolism not followed by the formation of an infarct. The lung area corresponding to the embolized arterial branch is ischemic, i.e., the zone is clarified and shows diminished or absent vascularization. This is more clearly evident in those cases with a congested lung. On the proximal side of this area the vascular pattern is well preserved. A progressive increase of the anemic area is generally indicative of a poor prognosis; central thrombotic extension is taking place. Oblique or lateral views are usually more informative than the routine anterior view. The same appearance of localized increased transparency occurs with bronchostenosis and associated valve-like mechanism. As far as areas of consolidation are concerned, attention is directed to certain diagnostic difficulties. First, one is not able to detect a definite area of consolidation until perhaps after the first 24 hours, or longer, following the onset of clinical symptoms. Second, the shadow of an accompanying pleural effusion may conceal the pulmonary lesion proper. Third, focal changes in congested lungs are not visualized too well. Fourth, intense pulmonary congestion and edema, in the presence of heart disease, need not be diffuse and hence may imitate pulmonary infarction. Since infarcts may involve any peripheral portion of the lung, including the surfaces of the interlobar fissures, it is imperative to obtain both oblique and at least one lateral view in addition to the routine anterior view. Failure to do so explains, in part, the unsatisfactory state of diagnosis heretofore. Grave illness of the patient, however, may require limitation to a single posterior view. Contrary to certain writings, infarct shadows are rarely triangular in shape, nor does the apex of a pyramidal shadow point towards the heart. Provided a tangential view is obtained—and hence the necessity of choosing a proper projection—the cardiac



FIGS 63, 64—33 yrs M History Rheumatic fever at age 17 Had six attacks of hemoptysis during the past 2 yrs He worked until very recently as a brick layer Was on digitalis Repeated hemoptyses occurred during the past 5 days Findings There was no fever, dyspnea, cyanosis and pallor were noted, there was no peripheral congestion. The heart was large, with a systolic precordial lift and a mitral stenosis murmur Auricular fibrillation was present, vr 83, pr 82, BP 112/78 There was dullness to percussion and marked diminution in breath sounds around the dorsal-caudal third of the rt lung with a few rales ventrally Rbc 38 mill, hb 10 gms, wbc 16800 with 87% polys

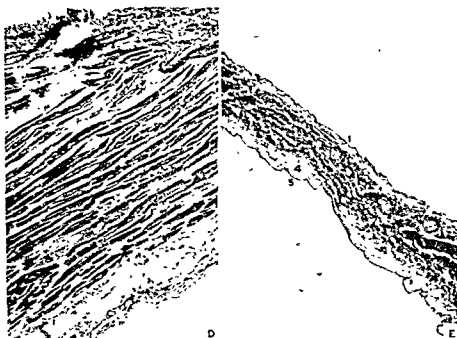
*Course* A profuse hemoptysis persisted 24 hrs later the patient was in vascular shock, BP 80/42 Rbc 28 mill, hb 8.5 gms wbc 41000 with 90% polys ECG. Auric fibrill, rt ax dev

*Roentgenograms* Post view Large heart of mitral configuration A large area of uniform density is noted which occupies the caudal area of the rt lung except a small area at the cardio-hepatic angle There is an increase in the vascular markings Lt post obl view The area of consolidation extends far dorsad

*Course* The profuse hemoptysis persisted, the anemia increased and the patient died the next day

*Postmortem* The heart weighed 550 gms The epicardial surface of both atria was granular and hemorrhagic There was a marked degree of calcific mitral stenosis, the aperture measuring but 1 cm in diameter There was a large firmly adherent thrombus in the left atrium The coronary orifices were patent There was atheroma of the pulmonary artery and its valves Microscopic Myocardial hypertrophy and degeneration The rt. pleural cavity contained 200 cm<sup>3</sup> of brownish fluid, and a fibrinous pleuritis was present dorsally and laterally The trachea and bronchi contained clotted blood The lt lung weighed 700 gms The rt lung weighed 1150 gms The arteries revealed many fatty atherosclerotic intimal plaques The rt lower lobe was dark red and completely solidified with the exception of a sharply delineated thin margin in the apical region The cut surface was of red-homogeneous appearance, not granular nor spongy The bronchioles contained red blood A bleeding point in the vascular tree could not be discovered Microscopic Infarction with superimposed interstitial pneumonitis

*Comment* The patient was afflicted by a severe degree of mitral stenosis, yet capable of doing strenuous work As so frequently happens, pulmonary infarction caused sudden aggravation and death The infarction of nearly a whole lobe is uncommon, and such severe and persistent hemoptysis resulting is apparently a great rarity

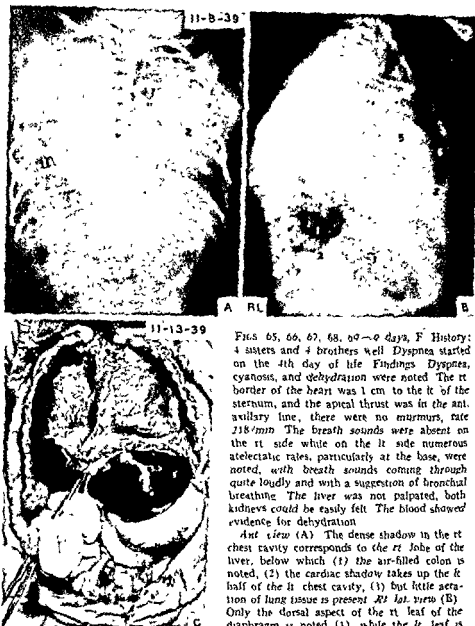


showed normal muscle development (microphotogram, D) The rt. leaf of the diaphragm showed an absence of the muscle layer 1, serosal lining, 2, blood vessel, 3, dense fibrous tissue, 4, loose fibrous tissue, 5, serosal lining (microphotogram, E).

*Comment:* This congenital malformation of the diaphragm led to such a diminution of the chest volume that death resulted from pulmonary atelectasis.

margin of an infarct shows a convex, slightly irregular, hump-shaped outline. Some of the shadows are roughly circular in shape and suggest a lung tumor or abscess. This may be caused by two or even more infarcts projected in a certain direction. Involvement of the lateral-caudal margins of the middle lobe will produce a long narrow shadow. Or one notes a hazy clouding at the base which partly or completely obscures the dome of the diaphragm. These findings must be differentiated from a small pleural effusion. The latter reveals a concave cranial border with density highest in the axilla, while an infarct does not produce such a picture unless, of course, it is complicated by an associated pleural effusion. Such a basal location is frequently associated with an elevation of the corresponding dome of the diaphragm, this is probably due to inhibition of the respiration and is much less frequently noted with other lung diseases associated with pleurisy. This abnormally elevated position of the diaphragm may not be visualized until the overlying density clears. Ill defined lobulizations are suggestive of multiple infarcts. The presence of normally aerated lung between multiple areas of peripheral consolidation is in favor of infarction and against a lesion due to bronchial obstruction. Haziness of infarct areas is caused by a surrounding reactive inflammatory zone or by an accompanying pleurisy. Resolution of an incomplete infarct shows as an area of reticular appearance that progressively clears entirely. Usually, however, cicatricial formation results in dense, band-shaped or narrow wedge-shaped





FIGS 65, 66, 67, 68, 69—0 days, F History: 4 sisters and 4 brothers well. Dyspnea started on the 4th day of life. Findings: Dyspnea, cyanosis, and dehydration were noted. The rt border of the heart was 1 cm to the lt of the sternum, and the apical thrust was in the ant. axillary line, there were no murmurs, rate 218/min. The breath sounds were absent on the rt side while on the lt side numerous atelectatic rales, particularly at the base, were noted, with breath sounds coming through quite loudly and with a suggestion of bronchial breathing. The liver was not palpated, both kidneys could be easily felt. The blood showed evidence for dehydration.

Ant view (A) The dense shadow in the rt chest cavity corresponds to the rt lobe of the liver, below which (1) the air-filled colon is noted, (2) the cardiac shadow takes up the lt half of the lt chest cavity, (3) but little aeration of lung tissue is present. Rt lat. view (B) Only the dorsal aspect of the rt leaf of the diaphragm is noted (1), while the lt leaf is seen almost in its entirety (2, 2), air is noted in

the stomach (3), and in the colon (4), the rt liver lobe (5), aerated lung tissue takes up the space between both the sternum and liver (6).

Course: Death 3 days later.

Postmortem The heart was displaced and weighed 30 gms. It was slightly enlarged but otherwise normal. The great vessels were normal. Both lungs were almost completely atelectatic. The liver weighed 125 gms. Its large rt lobe projected cranial far into the chest cavity but was separated from the chest contents by a thin serous membrane. This membrane represented the ventral portion of the rt leaf of the diaphragm while its dorsal portion contained muscle. The lt leaf of the diaphragm was normal in appearance and was muscular (picture in situ, C). Both phrenic nerves were grossly normal. All organs revealed congestion. Microscopic The lt. leaf of the diaphragm

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shadows. They are frequently seen at the base of the lung or perhaps an inch above the dome of the diaphragm. Bronchostenosis with secondary obstructive atelectasis causes an identical appearance.

**Disturbed Function of and Abnormal Findings Along the Diaphragm.** The optimum of the circulation cannot be maintained without a normal functioning diaphragm. Fluoroscopic examination has shown that the diaphragmatic excursions diminish or cease subsequent to a laparotomy; to this the postoperative depression of the circulation is partly attributed. Interesting experiments concerning the pre- and post-operative blood flow are cited in chapter XIV.

A diminished diaphragmatic excursion is observed in certain individuals with a long, flat chest and a low position of the diaphragm. Some of them reveal definite postural deficiency with increased lordosis of the lumbar region, marked torsion of the pelvis, poor abdominal muscle tone; signs and symptoms of marked vasomotor insufficiency are present in the upright position such as dizziness, tachycardia, fall in blood pressure; physical exertion occasions shortness of breath; and if, in addition, pain is found and perhaps a murmur, heart disease may be erroneously diagnosed.

There are cases where the chest wall is lifted craniad during inspiration while the diaphragm practically does not move. One may make the mistake of interpreting this relatively slight or apparently caudal movement of the diaphragm during inspiration as an actual one although it has not moved.

Left-sided relaxation of the diaphragm, due to either congenital muscular deficiency or to paralysis of the phrenic nerve, and left-sided herniation cause a considerable displacement of the cardiac silhouette. Following phrenic nerve interruption, the corresponding leaf of the diaphragm rises. The heart shifts towards the other side in about half the cases following left-sided operation, and in nearly all cases following a right-sided operation. In no instance does such a shift exceed 3.6 cm. A para-esophageal hernia may displace the heart ventrad and is occasionally related to attacks of retrosternal pain. A depression of the left diaphragmatic contour is visualized in the presence of a large left heart or when the pericardial cavity is markedly distended. The right diaphragmatic contour is seen depressed by a huge, aneurysmal dilatation of the left atrium. These changes in contour are best studied with the patient in the prone position.

Certain pulsatory observations should be added here. The fluid level in the stomach shows considerable motion in the presence of aortic regurgitation. Tricuspid regurgitation occasions a positive ventricular liver pulse which can be observed along the contour of the right leaf of the diaphragm.

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## CHAPTER VII

### CARDIOVASCULAR DISEASE FROM THE VIEWPOINT OF ETIOLOGY

**Avitaminosis.** It was thought until rather recently that the more severe forms of deficiency of vitamins B<sub>1</sub>, C and D revealed signs and symptoms referable to the cardiovascular system, but it now seems probable that the myocardial disturbances reported as accompanying C and D deficiency are caused by the simultaneous presence of B<sub>1</sub> deficiency, and it is with this in mind that the following paragraphs on rickets and scurvy should be read

**RICKETS (D avitaminosis).** There are two anatomical studies available. One author describes a moderate degree of enlargement of the left ventricle, with dilatation prevailing over hypertrophy and with the right heart participating when respiration was interfered with. No explanation has been offered for these findings. The other group of investigators refer to dilatation and hypertrophy of the right ventricle with only an occasional instance of associated left ventricular enlargement. Marked thoracic deformity and interference with respiration seem to account for these findings. Both thoracic and diaphragmatic respiration are impaired when the rigidity of the respiratory frame is lost. Interference with the expansion of the chest will interfere with the circulation. An actual loss of intrathoracic space during inspiration is observed in very severe cases. A deformity progressively develops whereby the heart is imprisoned in the space bounded in front by the sternum and cartilages and at the sides by the wall of costochondral enlargements which project into the cavity of the thorax. Atelectasis of the lungs is common.

The cardiac silhouette is found disproportionately large for the size of the chest and is accompanied at either side by longitudinal shadows with indefinite margins, produced by the atelectatic lung beneath the costochondral junctions; the latter add to the shadow as healing is accompanied by the deposition of lime salts. Scattered areas of atelectasis and chronic indurative pneumonia are noted in the lung fields.

An experienced pediatrician observed regression of the enlarged silhouette subsequent to treatment with Viosterol.

**SCURVY (C avitaminosis).** Right ventricular hypertrophy and dilatation of the heart was often found in young children who, after a prolonged course, died from this disease. The osseous changes in the ribs and the subperiosteal hemorrhages cause thoracic pain, the respiration is inhibited and this acts unfavorably on the circulation. Bronchitis and pneumonia may play an additional rôle. Cardiac enlargement, and occasionally pericardial effusion or hemorrhagic pericarditis, were found in adults. Roentgen studies apparently have not been published.

**BERIBERI (B<sub>1</sub> avitaminosis, thiamine hydrochloride deficiency).** Here a distended heart is seen, the lungs are normal, the thoracic cage is normal, the  
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genologic amplitude was associated with exactly the same alterations in the shape of the kymographic waves as referred to above. Proper nutrition with the addition of vitamin B<sub>1</sub> brought about a complete return to normal.

In the Occident severe thiamine hydrochloride deficiency occurs chiefly in alcoholics. It may also be observed in patients who are on a very restricted diet, or who suffer from chronic diarrhea or diabetes, or who have had surgical resections of the intestine. The clinical picture is not identical with the oriental beriberi, and this may be partly due to the fact that the majority of patients under consideration do not perform prolonged hard work. Roentgenologically the presence of pulmonary congestion has been mentioned but the descriptions from the Orient stress the predominance of right-sided failure. Dramatic improvements have been observed also in the occidental group accompanied by regression in the size of the silhouette following the use of vitamin B<sub>1</sub>.

**Disturbance of Metabolism and Internal Secretion. OBESITY.** This condition promotes cardiac impairment and occasionally may become the responsible factor for the development of cardiac failure. The clinical determination of heart size is extremely difficult. Two errors are likely to be committed. One is the assumption of right sided enlargement. The broad contact of the heart with the anterior chest wall, together with a shift to the right because of the absolute and usually relatively high position of the left leaf of the diaphragm, is responsible for this. The other error is the assumption of aortitis, the result of frequently finding an accentuated second aortic sound and systolic murmur. This is explained by a displacement of the ascending portion of the aorta ventrad and to the right.

Adiposity of the heart is understood to mean an increase in the amount of fat in the connective tissue located subepicardially, between the muscle bundles and muscle fibers and pericardially (periapically). The periapical fat layer may reach a thickness of 2.5 cm and a width of 5 cm and the epicardial layer may be as thick as 2 cm. It affects chiefly the right ventricular surface; its lines of demarcation against the cardiac muscle may be sharp or entirely obliterated.

The roentgenological study of the chest in obese individuals reveals a somewhat high position of the diaphragm and a broad contact of the cardiac and abdominal shadows. The vascular shadow is relatively broad and short and its right border is often quite straight and shows pulsations characteristic for the venous type. The right border of the silhouette is short. The appearance of the whole silhouette corresponds to the findings which are ordinarily noted during the phase of expiration or when a patient is examined in the recumbent position. A greater portion of the caudal heart border is visualized during inspiration and when the target is centered at a level more caudad than usual, i.e., over the ninth or tenth vertebral segment instead of the sixth. The increased width of the vascular shadow is occasioned by a greater spread of the aortic loop and an actual dilatation of the aorta may be easily excluded in the oblique views. The left lower pole region can seldom be distinguished clearly. Upon cursory examination, it appears to extend in a rather straight or slightly concave line toward the diaphragmatic contour. Fluoroscopy with a small shutter, preferably using radiation of greater wave length (low voltage), permits one to see the pulsating, convexly curved left lower pole within the

the Orient. The seriousness of the cardiovascular syndrome was and is probably due to the fact that it develops in people who perform strenuous work. The acute pernicious form is called Shoshin in Japan. The disease affects all age groups; breast fed infants acquire the disorder if the mother has a latent or very mild form of beriberi. Authorities on the tropical beriberi are not inclined to accept the diagnosis of beriberi heart unless it is associated with neuritic beriberi signs.

Anatomically one finds a marked enlargement of the right heart affecting especially the conus arteriosus, and dilatation of the intrapericardiac portion of the pulmonary artery and of both the central and peripheral veins. The left side of the heart, as a rule, shows no enlargement whatsoever. A pericardial effusion may be present but not necessarily so. There is a loss of tone in the heart muscle. The clinical picture is dominated by right sided failure, sometimes fulminating in type. The cause of this is thought to be primarily a dilatation of all the peripheral small arterioles resulting in a kind of diffuse arteriovenous anastomosis. These floodgates may be closed, with benefit to the patient, by the administration of pitressin.

The roentgenological appearance, in a well-advanced case, is characterized by a globular enlargement of the heart with a marked bulge of the right lower border and of the pulmonic arch which is displaced cranial and may reveal vigorous pulsation. The latter finding should be attributed either to a dynamic dilatation of the pulmonary artery or, what is more likely, to a prominence of the conus arteriosus. The vascular shadow often is enlarged to the right, indicating an enlarged superior vena cava. The pulmonary branches are not enlarged and there is no evidence of congestive failure in the lesser circulation. With the amelioration of the disease, the cardiovascular silhouette narrows and the pulmonic arch prominence disappears. Ricebran extract was shown to bring about rapid improvement of this condition. Following two intramuscular vitamin injections, the cardiac area in an infant diminished as much as 20% within two days. Regression of heart size is unquestioned. Often, however, it is not possible to compare the silhouettes correctly because the diaphragm descends as congestive failure disappears. Studies on the influence of pitressin medication on the size and shape of the silhouette should be of interest. Instructive facts have been obtained from roentgen-kymographic observations. A slight increase in the size of the amplitude of the heart shadow is noted during the early phases of the disease. As it advances, and as the heart enlarges and takes on a globular shape, an alteration is noted in the character of the pulsations. The normal shape of kymographic waves, as registered along the lower left silhouette border, resembles the shape of closed scissor, or a trapezium, or a mortise-chisel. But here the waves become rounded. As the disease and cardiac enlargement progress, the waves become shallow and broad. And during the Shoshin stage the amplitude is almost nil—less than 0.05 to 0.1 cm. These wave form alterations are thought to be quite characteristic, and their study will enable one to suspect the presence of a cardiac beriberi. With clinical im-

degree develops, and the increase in heart size and the diminution of roent-

cardiac enlargement do not necessarily run parallel. The clinical finding of a booming first sound, thrill, split sounds and auricular fibrillation often necessitates the differential diagnosis between this condition and mitral stenosis. The clinical evaluation of the heart size in the presence of thyrotoxicosis is quite difficult because the forcible heart action easily leads to a false impression of enlargement.

Before ascribing cardiac enlargement to thyrotoxicosis, it must be taken into consideration that it may be accounted for by associated cardiovascular disease. This, perhaps, may be responsible in part for the fact that the number of enlarged hearts increases with age. However, the duration of the process may be a deciding factor. Certainly in cases with gross cardiac enlargement, other coincidental cardiovascular disease should be suspected. If enlargement is present and due only to the thyrotoxic factor, it is generally of only slight or moderate degree. The incidence, obviously, varies with the type of material. In four publications, each dealing with a series of not less than 125 and not more than 200 cases, an enlargement is referred to in 44%, 45%, 46% and 47% respectively. Another author insists upon a figure of 83% for 80 cases but in this group a marked filling out of the waist of the heart was considered as a criterion of enlargement. Most of these patients were hospitalized and the figures would be far lower if milder cases were also included. In a recent series of 102 noncomplicated thyrotoxic cases, 26% showed the cardiac area to exceed the normal limits. All instances in which auricular fibrillation is established for at least a few months' duration and cases with failure show enlargement, while paroxysmal attacks of fibrillation are easily compatible with a normal size. No enlargement should be expected when the symptoms are mild and of shorter duration. The form of the cardiac silhouette may be globular with a widening either to the left or to both sides. It is hardly possible to decide roentgenologically which ventricle is more affected. The left atrium is usually not enlarged, or at least not out of proportion to other chambers of the heart, high degrees of enlargement are not observed.

A marked bulging along the upper aspect of the left cardiac contour, corresponding to the conus arteriosus, is not noted. More craniad, however, the pulmonic arch is quite often prominent, independent of any other change in heart size. This prominence may either appear as a convexity or merely render the left profile straight, it is sometimes concealed within the shadow of the upper portion of the descending aorta or of a prominent upper ventricular contour as in the case of cardiac enlargement. It can be better visualized by rotation toward the right anterior oblique view. It is more often recorded on orthodiagrams than on films with short exposure, since in the latter case the phase of diastolic collapse often will be registered. The length of the arch is frequently increased, its average for 51 cases having been determined as 5.6 cm. These changes in the pulmonic arch are often visible at an early stage of the disease and quite marked with higher degrees of toxicity. The shape of the total silhouette has been described as having a resemblance to a ham. The hilar vessels retain their normal size. It is remarkable that even in the presence of failure, the roentgenological signs of lung congestion (see chapter VI) are but little marked.

The aortic arch is found to reach far craniad in a number of instances but



non-pulsating, less dense, triangular shadow that consists of extrapericardial fat and the ligamentous fold extending between pericardium, mediastinal and diaphragmatic pleurae. Ordinary chest films, as a rule, do not permit of this differentiation because of the use of higher voltage and insufficient exposure, and because of the lack of aid which is offered by the study of pulsations. Fluoroscopy of the chest of obese individuals causes overestimation of the heart size because of the increased object-screen distance. Orthodiagrams or

telerontgenograms are, therefore, of importance. Another feature in fluoroscopy is a certain haziness of all contours. This is caused by the increased amount of secondary radiation

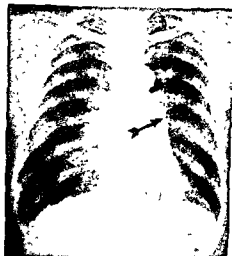


FIG 70—55 yrs, F Palpitation and weakness since age 51 Weight loss and precordial pain at effort since age 54 Nodular thyroid gland Cardiac hyperactivity, tachycardia First apical sound booming, harsh systolic murmur and faint thrill over pulmonic area, B.P. 130/60 ECG essentially normal BMR +40% Thyrotoxicosis Ant view absence of cardiac enlargement Marked

#### clinical signs.

probably arising from aberrant thyroid tissue in the mediastinum For further remarks see tracheobronchial stenosis (this chapter).

**THYROTOXICOSIS.** The roentgenological appearance of shape and activity is, in many instances, sufficiently characteristic, especially with marked degrees of toxicity. It is helpful, therefore, in determining causes of obscure cardiac symptoms or enlargement, with or without failure. It may actually lead the clinician to suspect goiterous etiology. But even with the established clinical diagnosis, the knowledge of the exact degree of enlargement and hyperactivity aids in judging the severity of the lesion. However, severity of the disease and

It should be possible, theoretically, to visualize the epicardial fat layer provided it is large enough and sufficiently well demarcated from the cardiac muscle. A tangential view of the right ventricular region should be more valuable than the straight anterior view. Requirements for this are: short exposure, good contrast (rotating grid-diaphragm), good definition (fine focal spot) and an exposure to penetrate the mass of the heart satisfactorily. This requires a multiple of the time used with the ordinary standard chest technique

**GOITER.** It occurs in the newborn in endemic goiter regions and is found to be accompanied by heart weights exceeding the average figures. This fact makes it very probable that the roentgenological observation of enlarged silhouette areas was correct and not merely caused by thymic enlargement Right-sided cardiac enlargement was simulated in one rare case by a large intrathoracic goiter,

cardiac enlargement do not necessarily run parallel. The clinical finding of a booming first sound, thrill, split sounds and auricular fibrillation often necessitates the differential diagnosis between this condition and mitral stenosis. The clinical evaluation of the heart size in the presence of thyrotoxicosis is quite difficult because the forcible heart action easily leads to a false impression of enlargement.

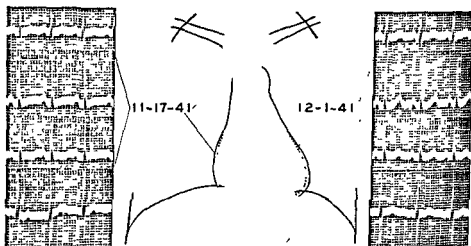
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The aortic arch is found to reach far craniad in a number of instances but

the aortic knob is not unduly small. It is not especially prominent unless made so by displacement from a substernal goiter. Often the superior vena cava is quite prominent. This finding is independent of congestive failure.

The hypertrophy and enlargement is accounted for partly by a direct toxic effect on the muscle cells and partly by the increased amount of work. The circulating blood volume, the minute and stroke volume and the blood velocity



of pregnancy, the wt. was 709 kg enlargement of the thyroid gland 5 in.). A typical thyrotoxicosis was aud Bmr plus 55% ECG · Sr, rate 126/min, T deflections of low voltage

*Orthodiagram.* Minimal enlargement of the silhouette. Fluoroscopy showed overactive pulsations.

*Course:* The patient was kept on Lugol's solution for a period of 10 days. The ECG. then showed a rate of 90/min and the voltage of the T deflections had increased. Fluoroscopy showed the pulsations less overactive, and in the orthodiagram the silhouette was a trifle smaller. The next day a subtotal thyroidectomy was performed. Microscopic. Diffuse toxic hyperplasia, exophthalmic type. One week later the bmr. was plus 12%, the E.C.G. showed a rate of 100/min. and the T deflections of normal voltage

*Orthodiagram:* Normal sized silhouette, smaller as compared with the findings 2 wks ago

*Comment:* The heart of this thyrotoxic patient already showed improvement under the influence of the preoperative iodine treatment, as revealed by both the roentgenologic and electrocardiographic study. Further progress was noted one week postoperatively, at which time the heart unquestionably had receded in size and the abnormal findings in the E.C.G. had disappeared

are increased. The lumen of the pulmonary artery in 6 cases was found at necropsy to be 17% larger than the aorta, but the expected finding for this case was plus 80%. Another investigator reported the lumen of the pulmonary artery to be 56% above the average. These findings are unusual in type, "flabby" or hypotonic. They are sometimes increased in amplitude along the

cardiac silhouette and are conspicuous along the pulmonic arch, aortic knob, ascending aorta and superior vena cava, as observed in the different views. Clinically, one notices a marked increase in the jugular pulse as an expression of the increase in blood velocity. The adjacent intrapulmonary shadows likewise reveal an increase in their movements. Kymographic tracings of the left lower ventricular contour reveal a deviation from the normal graphic ap-



FIGS. 72, 73—66 yrs, F Goutier since age 31 Partial thyroidectomy age 41. Nervousness, weakness, palpitation since age 44 Slight degree of failure and irregular heart action since age 64. Cardiac enlargement and hyperactivity, booming first sound,  $P_2$  accentuated, rhythm irregular, rate 116 Tibial edema, slight B.P. 160/80 ECG: auricular fibrillation,  $T_{1,2}$  positive, lt axis dev Bmr  $+12\%$  Toxic adenoma Ant view: moderate enlargement of silhouette L 15.2, B 11.5, T 15.8,  $T_D$  11.0 cm (on 7 foot film) Slight prominence pulmonary conus (bl arrow) Descending thoracic aorta prominent (wh arrow). Substernal goiter with displacement of trachea to rt. No congestive lung failure. Lat view (illustration omitted). Lt atrium slightly prominent, descending thoracic aorta elongated. Course removal of thyroid, leaving only 1/40 of tissue Microscopic nodular hyperplasia. Follow up study two years later feels well, gained 10 kg in weight Apical thrust farther in; no edema, no hyperactivity, heart sounds normal without basal accentuation, rhythm regular, rate 82 B.P. 145/90 ECG: n.s.r., a-v conduction time 0.22 sec,  $T_{1,2}$  positive, lt axis dev Bmr  $-26\%$  Ant view normal sized silhouette L 14.0, B 10.2, T 12.6,  $T_D$  10.7 cm (on 7 foot film). Vascular shadow unchanged. Trachea not displaced Lat. view (illustration omitted). Lt atrium not enlarged Rib pattern and diaphragm position are practically exactly comparable in the two films Definite regression in cardiac size has occurred

pearance. The diastolic rise is rather steep without formation of a plateau and the duration of the medial movement is quicker and greater in extent than in the normal heart beating at approximately the same rate; the plateau formation of the systolic stage is almost absent Crests and troughs somewhat resemble a sinus curve. How far hypotonicity of the myocardium may account for these changes remains to be investigated.

The roentgenological differentiation between the hearts in thyrotoxic and mitral valvular disease is simple, especially when the thyrotoxicosis is in an active phase. The mitral heart is lacking in hyperactive pulsations and they may even be absent in the lower pole region. The middle portion of the left contour, the conus region, is the most prominent area. The aortic knob is quite small, sometimes invisible. The enlargement of the left atrium with the consequent displacement of the esophagus is marked and the lung fields reveal a more or less characteristic appearance as described in chapter VI.

The preoperative period, with rest in bed and iodine medication, is an important part of the treatment because it is then that cardiac enlargement may be reduced. During this period the basal metabolic rate, the pulse rate and the pulse pressure are observed to diminish, and auricular fibrillation may occasionally change to a normal sinus rhythm. It has even been stated that the proportion of hearts reduced in size as the result of the whole treatment is no greater than that resulting from the preliminary bed rest and iodine medication.

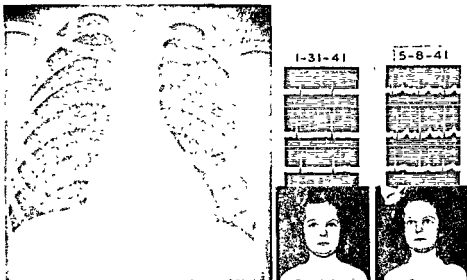
With the improvement of the condition, after operation or irradiation, the pulsations are seen to return to a normal aspect. The prominence of the pulmonic arch likewise disappears in about three-fourths of all cases within a period of a few months. The cardiac shadow which has been normal in size remains unchanged or may slightly increase with a gain in body weight. Whether this increase corresponds to an actual volumetric increase or is only apparent, is not known. Perhaps where marked wasting with some anemia has existed previously, there is an actual increase but it should be recalled that an increase in body weight tends to raise the diaphragm and so to increase certain diameters. Where congestive failure with auricular fibrillation has existed, marked reductions in size have been noted but it is almost impossible to compare correctly roentgenograms of the same individual taken during and after the stage of congestive failure. Where enlargement often associated with auricular fibrillation was present, a diminution certainly may take place but, as far as we know, does not necessarily do so. This problem is complicated because associated cardiovascular disease has not always been sufficiently considered from the statistical aspect and because an exact roentgenological technique has been adhered to in only one study. An increase in the size of the silhouette at some time following thyroidectomy might point to the development of a postoperative myxedema.

**MYXEDEMA.** An enlargement of the silhouette occurs but is not a necessary accompaniment. The enlargement is bilateral and symmetrical. With lesser degrees of myxedema the enlargement is less marked.

The pulsation is sluggish and the presence of a large silhouette in the lower pole and hydrothorax are rarely observed by associated cardiovascular disease. The widening of the silhouette is caused either by cardiac dilatation or by pericardial effusion or is due to a combination of both factors. Which factor plays the more predominant rôle is not yet known but it seems that effusion is

more often present than is suspected. In three instances its presence was proven by paracentesis.

After thyroid medication the size of the silhouette usually undergoes a slow



FIGS 74, 75—9 yrs F History Headache, projectile vomiting, preference for warm weather, fatigue and drowsiness, visual disturbance, constipation, an undue gain in wt. during the past year Findings Ht 127 cm (50"), wt 27.4 kg (60½ lb) The reaction pattern was slow The skin was cool, coarse, and dry There was pallor of the mucous membranes The face was puffed Examination of the heart revealed nothing significant The blood showed hb 11 gms, rbc 3.8 mill, cholesterol 584 mgm % The b.m.r. was minus 36% Other laboratory studies were of no significance ECG Sr with an occasional premature auricular beat, rate 72/min, P deflections of low voltage, initial deflection with maximal voltage of 8½ mm, T negative in leads I and II, isoelectric in III

*Ant view.* Cardiovascular silhouette normal as to size, shape and position

*Course.* A subsequent study showed the BP 90/60, and the b.m.r. minus 42%, 47% The visual fields were normal The child was placed on thyroid extract, gradually increasing from 0.03 gms (grs ½) to 0.12 gms (grs II) daily Within several weeks the child lost the symptoms referred to above The cholesterol was 168 mgm %, and the b.m.r. was minus 5%, 2% ECG Sr with sinus arrhythmia, rate 90/min The voltage of P and QRS increased, the latter now maximal 12 mm T positive in the three leads

*Comment.* An instance of juvenile myxedema associated with normal cardio-roentgenologic findings If, in the presence of myxedema, the silhouette is found to be enlarged, one should consider the possibility of pericardial fluid If actual cardiac enlargement is proven to be present in association with hypothyroidism, one should investigate the duration of the latter and consider the possibility that it may have acted on an already damaged heart muscle

regression The minimum, as a rule, is reached during the course of a few months The greater the degree of enlargement, the greater the amount of reduction If this is marked, resorption of fluid is most apt to account for it, at least in part

Total ablation of the thyroid gland is followed by myxedema The silhouettes

of those patients who had congestive failure before operation show variations in changes which seem to be the result of two opposing factors; first, the effect of the hypothyroid state tending to increase the size of the silhouette, and second, the restoration of circulatory compensation tending to decrease the



FIGS 76, 77, 78—45 yrs, F. Typical myxedema. (a) Before therapy B m r. —32% (b) 16 days later. Patient received 78 grs Armour's thyroid Silhouette diminished in size B m r —4% (c) 6 months later After additional 190 grs. Armour's thyroid b m r —3%. Silhouette considerably further decreased. Courtesy Dr George W Holmes, Dr James H Means and Dr. J. Lerman Mass Gen'l Hosp, Boston.



size of the silhouette. The difficulty, however, in comparing this size during treatment and failure respectively should be borne in mind. Cases without enlargement of the silhouette.

**ACROMEGALY.** The heart is found to be considerably enlarged as a partial expression of the splanchnomegaly. Cardiac hypertrophy and dilatation were

produced experimentally in dogs by feeding anterior pituitary extracts. The average heart weight was 380 grams as compared with 180 grams of the control animals.

The cases which have been reported had acromegaly over a period of many years. A few roentgenological observations indicate that with shorter duration the silhouette is not necessarily enlarged.

**Disturbance of Arterial Blood Pressure, of Volume and Constituents of the Blood. GLOMERULONEPHRITIS.** a) *Acute.* Postmortem studies have demonstrated that in a certain percentage of cases, dilatation of the heart occurs as early as the first or second week of the disease, and hypertrophy even during the second or third week. Clinically there may also be present dyspnea, slight cyanosis, cough, crepitant rales, gallop rhythm, systolic murmurs, and E.C.G. evidence of left ventricular strain.

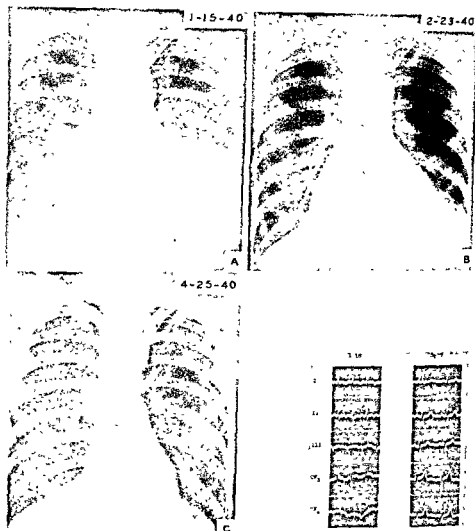
Rarely will one have at one's disposal the roentgen findings of the patient's cardiovascular silhouette prior to the onset of the illness. But in comparing one's observation with those of a few weeks later, when clinical and laboratory findings are normal again, one notices that an enlargement of a minor degree is commonly associated with the acute phase of the disease. There is an increase in the blood pressure and most probably an increase in the amount of circulating blood (prevalence of plasma over blood cell volume). Some widening of the pedicle with an increased prominence of the pulmonic arch is also occasionally found. An enlargement of the silhouette may be due to the heart proper and this may reasonably be assumed to be the case in the absence of peripheral edema, pleural fluid or pulmonary edema. In many instances, however, an accumulation of pericardial fluid is responsible for the enlargement of the silhouette and this may be safely diagnosed to be the case in the presence of peripheral edema, pleural fluid or pulmonary edema. Another point deserves consideration. Acute nephritis is usually associated with a relatively high position of the diaphragm. Water retention in the abdominal viscera and cavity is responsible for it. This may or may not be striking, and may not be recognized until the renal process is subsiding with the accompaniment of associated wide-spread capillary damage and disturbance of the blood chemistry. The shape of the silhouette will be influenced, of course, by the position of the diaphragm.

The anuric state is accompanied by a peculiar appearance of the lung fields (described in chapter VI), which is accounted for by interalveolar transudation, particularly affecting the central portions of the lung. A diminished transparency of the lung fields may also be found in three additional instances which can well be found in combination: a moderate degree of congestive lung failure of the common type, pleural transudation, and edema of the soft tissues of the chest wall.

b) *Subacute and chronic* The first roentgenological sign of left ventricular hypertrophy appears apparently in the form of a slight bulging in the middle portion of the left lower contour and precedes the caudal displacement of the lower pole region. Sufficient statistical data are not at hand to permit a statement as to how much time must elapse until the hypertrophy becomes recognizable roentgenologically.

In acute nephritis the heart seems to dilate primarily, while in essential





**FIGS 79, 80, 81, 82**—38 yrs, M History 2 wks after a severe sore throat, the urinary output diminished A few days later ankle edema and dyspnea appeared Findings (1-13) Low grade fever, slight degree of cyanosis and dyspnea, pretibial and sacral edema, pleural effusion—lt more than rt Apical thrust forceful, 14 cm to lt of midline in 5th sp BP 156/90 Hb 10 gms, rbc 3.2 mill, wbc 8150, urea n 23 mgm%, total protein 5.63, alb 3.56, glob 2.07 gms in 100 cc, a/g ratio 1.7,  $\text{CO}_2$  combining power 49 volumes% Cloud of albumin, numerous casts and rbc's in urine Hemolytic and viridans streptococci in throat

**Course** 1000 cc of fluid were removed from the lt pleural cavity

**Ant View (1-15, A)** Fluid is noted at both bases, more on the lt The central lung fields show a diminished transparency, but the peripheral and cranial portions appear normal, the vascular pattern is not increased The size of the cardiac silhouette is difficult to evaluate; apparently there is little enlargement The diaphragm is unduly high

... was done at the lt side, yielding  
100 diastolic The tempera-  
The patient became restless,

hypertension hypertrophy is thought by some to be the first response. No essential anatomical differences are noted for the heart in the later and advanced stages of either condition. The average lower blood pressure and shorter dura-

the neck veins were engorged and cyanosis was present, the lungs showed moist rales, a marked dullness to percussion over the lower half of the sternum was noted with normal findings over the upper half, the heart rate was increased and a very pronounced gallop was heard. *Fundi*: Slight degree of retinal edema, a few superficial hemorrhages and an occasional spot of cotton-wool exudate, no evidence of arteriosclerosis. *E.C.G.* (1-18): N.s.r., rate 135/min, normal P and P-R, initial deflection shows lt axis dev, maximal voltage in limb leads  $6\frac{1}{2}$  mm, S-T<sub>1</sub> arched, T<sub>1</sub> neg, T<sub>2,3</sub> pos, T in CF<sub>1,2</sub> plus-minus. In the course of 18 hours patient was given 0.03 gm (grs  $\frac{1}{2}$ ) of morphine sulph. s.c., 12 cat units of digalen i.m., 0.8 gm (grs XII) of aminophyllin i.v., and oxygen inhalation, and patient improved. Findings: A marked degree of gallop still persisted. B.P. between 150 and 165 systolic, 98 and 108 diastolic. Hb 10 gms, r.b.c. 3.7 mill, w.b.c. 20100, urea n 75 mgm %, creatinin 15 mgm %. Patient was started on sulfapyridine 1 gm (grs XV) q 4 hrs for 6 days, and tid for subsequent wk. Within 24 hrs the fever disappeared, the gallop disappeared simultaneously and the patient felt better. A small blood transfusion caused appearance of Cheyne-Stokes respiration, pulmonary rales and a rate of 160/min, with morphine and aminophyllin bringing relief. Gradual and steady improvement then followed. From 1-23 to 2-14 the urea n fell from 80 to 13 mgm %. Albumin, casts and r.b.c.'s persisted in the urine. Treatment was bed rest, low-salt and high-protein diet. Digitalis was discontinued on 2-13.

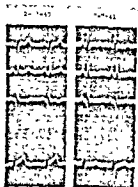
*Ant View* (2-23, B). There is fluid at the lt base. The lung fields reveal normal transparency. The vascular pattern is slightly increased. The cardiac silhouette is enlarged, with the contours revealing no subdivision (most probably pericardial effusion).

*Course*: The retinitis cleared completely. Findings: B.P. 166/115. Fluoroscopy (3-27): Entirely normal appearance of chest and cardiovascular silhouette, oblique diameter, orthodiagraphic, 14.2 cm. *E.C.G.* (3-27): N.s.r., rate 72/min, normal P and P-R, initial deflection with lt axis dev and normal voltage, normal S-T segments, T positive in I, II, III, CF<sub>1,2</sub>. One month later, the B.P. had reached a normal level. The urine still revealed abnormal findings when the patient exerted himself.

*Ant View* (4-25, C). Normal appearance of chest and cardiovascular silhouette.

*Course*: 5 mos. later there was an almost complete recovery. The B.P. was 130/95. The urine was free of albumin but showed an occasional rare cast, and a positive reaction to occult blood persisted. The urea n was 18 mgm %, and the urea clearance was 80%.

*Comment*: A Clinical. Left ventricular failure commonly accompanies acute glomerulonephritis. The outstanding diagnostic features are: Gallop and pulmonary congestion (dyspnea). In this case two interesting facts were demonstrated. First, the untoward effect of intravenous fluid (blood). Second, the unfavorable effect of infection, for no sooner had sulfapyridine become effective than both the fever and gallop disappeared. B. Electrocardiographic. The first tracing, which should be compared with the second one, revealed the pattern of acute left ventricular strain, i.e., a negative T in lead I. The low voltage was due to water retention in the body tissues and cavities. C. Radiologic. The first roentgenogram shows evidence of water retention. High position of the diaphragm, fluid chiefly in the left pleural cavity (this is rather characteristically noted with left ventricular failure provided sinus rhythm is present), and central, interstitial- and perhaps also parenchymal-edema. Note absence of congestion in the peripheral lung fields. The second roentgenogram reveals the presence of pleural fluid exclusively at left and, most probably, fluid in the pericardial cavity. The diaphragm has not yet reached its normal position. This is evident by inspecting the third roentgenogram, which was exposed at the end of quiet expiration. Note here the normal size of the heart. Courtesy of Dr. Edward Weiss, Temple University Hospital, Philadelphia, Pa.



FIGS. 83, 84, 85.—56 and 61 years, M. History: Malaria. Cholecystectomy at age 42. When seen first he had suffered for two years from mild effort dyspnea, abdominal discomfort and weight loss. Findings: There was no evidence of failure. The liver and spleen were slightly enlarged. The heart was in vertical position and showed a moderate degree of enlargement to the left. The B.P. varied at different times between normal and 155/110. The urine showed albumin. The blood showed normal n.p.n. and proteins, hb. 14.5 gms, r.b.c. 5 mill, w.b.c. 24650 with 86% polys. No evidence for parasites. Wassermann test negative. ECG: Minimal notching of P, otherwise nor-



mal. Repeated follow-up studies for a period of 4 yrs. showed no particular changes. The slight enlargement of liver and spleen and the leucocytosis persisted. The sedimentation test was 9 mm. and there was no fever at any time. The B.P. readings showed elevation at times. Fundi: Minimal amount of sclerosis, no retinitis. ECG (2-21-40): Essentially same as previously. Subsequently the patient lost in weight and complained of muscle cramps. Findings: Wt. 56.4 kg. (124 lbs.). The clinical picture was one of uremia. There was no evidence for heart failure. The heart was considerably enlarged, rate 80/min. B.P. 180/110. The urine showed much albumin, its specific gravity varied between 1008 and 1009. The urea clearance was between 5 and 10% of the normal. Hb. 9 gms, r.b.c. 3.3 mill, w.b.c. 28250 with 91% polys. Urea N 128 mgm %, creatinine 13.4 mgm %, CO<sub>2</sub> combining power 26 vol.%, proteins 6.65 grams with an a/g ratio of 1.5, calcium 7.6 mgm %, phosphorus 12 mgm %. Fundi: Marked atherosclerosis of choroidal vessels, attenuation of peripheral retinal arterioles. ECG (7-7-41): As compared with the previous tracing two changes had occurred: first, a marked lowering of the voltage of T in all leads; second, a lengthening of the Q-T interval.

*Roentgenogram, post view (7-17-41).* The heart is generally enlarged but more so on the left. The lung fields are of normal appearance. Left basal adhesion noted.

*Course:* The clinical picture was one of progressive, quiet uremia. On 8-19 an occasional hemoptysis occurred, and a few days later edema of the legs developed. The wt. was 60 kg. (131 lb.). The respiration was somewhat deepened but there was no dyspnea. A few rales were heard anteriorly. Roentgenogram (8-25): Definite changes were noted

tion of life in nephritics lead, as a whole, to an average lesser degree of hypertrophy and dilatation. The level and the duration of the blood pressure elevation represent only one factor influencing the size of the heart, i.e., chiefly the left ventricle. Other factors to be considered are the constitutional capacity to develop cardiac hypertrophy; and the damage resulting from physical overexertion, emotional strain or excess fluid intake. Nor is there a strict correlation between heart size and renal failure. Cardiac enlargement is frequently, but not regularly, met with in the final stage of chronic nephritis and pyelonephritis. Roentgenologic evidence of left heart failure is noted if the clinical picture ends with heart failure, but this is not the common course of events. The left atrium is then also enlarged. The lung fields during the final stage may

spi  
mc

dilatation. Roentgen studies on this type of case have not been reported.

**ESSENTIAL VASCULAR HYPERTENSION.** One cannot expect a characteristic appearance of the cardiovascular silhouette. Here some of the influencing fac-

in the lung fields and they were the same as observed later. The degree of anemia increased, hb 5 gms, r.b.c. 1.9 mill. Pulmonary rales continued off and on and were of irregular distribution.

*Roentgenogram, post. view.* (9-5-41): Small patchy densities of irregular distribution are noted. The diaphragm is higher than previously.

*Course:* At no time was there fever, nor did cardiac failure appear at any time. The urea N rose to 165 mgm %, and the CO<sub>2</sub> combining power was 14 vol %. Death (one week following the last roentgenogram).

*Postmortem:* The weight of the heart was 575 gms. There was present dilatation and a mild degree of hypertrophy. There were pale-yellowish areas in the myocardium. The valves and coronary arteries were normal. Microscopic: Uremic myocardial degeneration. The left pleural cavity was obliterated. The weight of each lung was 325 gms. All parts were well aerated. The cut surface showed everywhere not very recent hemorrhages which had begun to diffuse out into the lung tissue. They measured about 0.5 cm. in diameter and averaged about one to two to the square inch of cut surface. Microscopic: Emphysema, patchy macular hemorrhages, chronic interstitial pneumonitis. The kidneys showed a third stage of diffuse glomerulonephritis. The liver weighed 1400 gms, and the spleen was enlarged.

*Comment:* The patient suffered from chronic glomerulonephritis that led to renal failure. The change in the roentgenologic appearance of the lung fields was due to the widespread appearance of patchy macular hemorrhages and interstitial pneumonitis, both undoubtedly sequelae to the disturbed chemistry of the body tissues. Neither congestion nor infarction accounted for these findings. The position of the diaphragm in the two roentgenograms is not identical. Anticipating an aggravation of the patient's condition, the technician had been requested to take the first roentgenogram with the patient in recumbency and to obtain the exposure at the end of tidal respiration. However, an exposure at the end of forced inspiration was obtained. When the second roentgenogram was obtained the patient was semicomatose, and cooperation in the sense of forced inspiration, in order to duplicate the former condition, was impossible. The enlargement of the heart was presumably due to myocardial disease on the basis of prolonged renal failure and hypertension. The appearance of ECG changes indicated myocardial dysfunction and a lowering of the blood calcium—both on the basis of severe renal failure.

tors are: the type of hypertension, permanent or transitory, affecting the systolic level only or both systolic and diastolic levels; the degree and duration of the hypertension; the age and constitutional type of the patient; or complicating factors such as coronary artery disease, anemia, aortitis, valvular disease, and presence or absence of failure. An increased prominence of the aortic shadow, especially in the region of the knob, and of the descending aorta is often present but may quite as well occur with atherosclerotic dilatation or merely in connection with more advanced age.

The aortic shadow appears in all views to be moderately and uniformly di-



FIGS. 86, 87—12 yrs, F Headache, weakness, dyspnea on exertion Weight 24 kg, height 142 cm Apical thrust heaving, slightly displaced. A<sub>2</sub> much accentuated B.P. 235/160 Kidneys show low concentrating ability Blood urea n rising from 13 to 120 ECG: Lt axis dev. Malignant hypertension and nephrosclerosis *Ant view* Lt ventricular silhouette is prominent to lt and caudad (bl arrow) Aortic shadow is moderately widened and lateral contour of distal portion of ascending aorta (wh arrow) is in a line with contour of rt. cardiac border *Lt ant obl view* Prominence and rounding of lt ventricular contour (bl arrow) Prominence of ventral aortic contour (wh arrow) Dynamic dilatation of aorta. Postmortem Marked hypertrophy of lt. ventricle, average thickness of wall 2.4 cm Aorta small, circumference 4 cm

lated when young individuals are affected either by primary malignant or secondary nephritic hypertensive disease, especially if a high diastolic pressure is present. Here the internal pressure is the sole cause of dilatation, the post-

of the aortic s  
pears slightly  
of the left clc  
prominent. In the left anterior oblique view, the shadow of the second portion

of the arch may overlap dorsally the shadow of the spine. A cylindrical dilatation with a more marked prominence in the region of the ascending aorta renders the presence of syphilitic aortitis probable. There is no doubt, however, that marked and prolonged hypertension in the adult is compatible with a normal roentgenological appearance of the aortic shadow. The degree of the pulsatory amplitude is likewise not uniform. The presence of a wide pulse pressure tends to be combined with larger excursions, but these tend to become diminished when the diastolic pressure is high and during the stage of failure. Often, especially by observation in the oblique views, it becomes obvious that we are

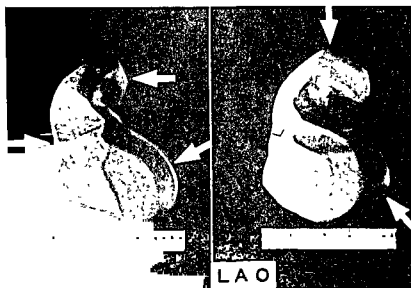


FIG 88—Old man. Hypertensive and atherosclerotic cardiovascular disease. Roentgenologic, volumetric reconstruction. *Ant view*: marked enlargement of lt ventricle caudad and to lt (lt lower arrow). Caudal shift of intersection between rt vascular and cardiac border (rt arrow), caused by enlargement of ascending aorta. Distal portion of arch (lt upper arrow). *Lt ant obl view*: prominence of lt ventricle dorsad (lt arrow). Enlargement and wide swing of aortic arch (upper arrow). Courtesy Prof Dr G G Palmieri, Bologna, Italy.

dealing, in the main, with a displacement of the aortic shadow in toto rather than with a marked expansile change. There is commonly noted in well compensated cases an increased amplitude of the left auricular appendage.

Provided the hypertensive stage has endured for a considerable period, the cardiac silhouette is usually slightly enlarged. This enlargement is to the left, caudad and dorsad exclusively. The left lower contour is elliptical with the main bulge craniad to the left lower pole region. L increases proportionately more than T. Occasionally one meets with a case normal in appearance despite long standing and marked hypertension and the ordinary diameters may be within the normal limits in rather a large percentage. The silhouette of some patients progresses very slowly indeed, sometimes only a few mm over years; it seems reasonable to assume that in these cases very little coronary artery dis-

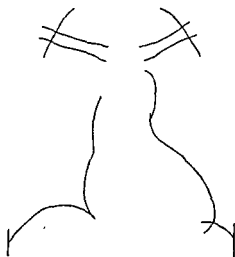


FIG. 89—25 yrs, F. Recurrent headache and epistaxis. Apical thrust resistant  $A_2$  accentuated. B P. 240/120. Blood urea increased. *Ant. view*: aortic configuration, marked elongation of lt lower contour, L 151, B 10.3, T 13.9, Th 24.4 cm. Aortic shadow slightly prominent at either side. *Rt. ant. obl. view* (illustration omitted): lt. atrium not enlarged. Death due to cerebral hemorrhage during sexual intercourse. Postmortem: marked hypertrophy, moderate dilatation lt. ventricle. Arterio- and arteriosclerosis of kidneys. Aorta small, without intimal changes.

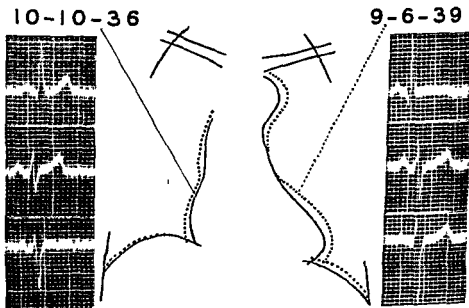


FIG. 90.—59 and 62 yrs, M. History. The patient was studied for other than signs or symptoms referable to the c-v system. Findings (age 59) There was no failure,  $A_2$  was accentuated, the B P. was 180/120. Fundi: Grade I arteriosclerosis, hypertensive type, and grade I choroidal sclerosis. ECG: S r with premature auricular beats, lt. axis dev,  $T_{1,2}$  positive.

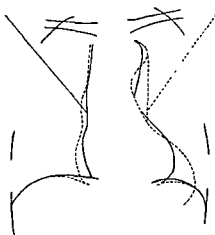
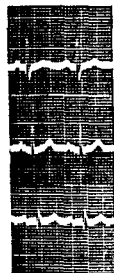
*Orthodiagram*: Very slight elongation of the lt ventricular area. Fluoroscopy showed the aortic shadow of increased density.

*Course*: Three yrs later again complaints not referable to the c-v system, but also headaches. Findings (age 62): There was no failure, the heart was slightly enlarged and  $A_2$  accentuated, the B P. was 200/130. Fundi: Grade I plus arteriosclerosis, hypertensive type, and grade III choroidal sclerosis. ECG: S r with premature auricular beats, lt axis dev,  $T_1$  nearly on base line,  $T_{2,3}$  positive.

*Orthodiagram*: As compared with the previous study, a moderate increase in heart size and a more marked prominence of the aorta are noted.

*Comment*: The hypertensive condition was well tolerated but its continued effect expressed itself in alterations of both the roentgenologic and electrocardiographic findings.

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FIG. 91—44 and 51 yrs., M. History This high strung and emotionally unstable patient was aware of high blood pressure for the past 10 yrs. There was no appreciable effort limitation. Findings. The heart was not enlarged, there was an apical systolic murmur of minimal degree, not heard over the aorta.  $A_2$  was accentuated. There was no gallop. B.P.: Systolic from 126 to 180, diastolic from 93 to 142. Wassermann negative. ECG: Normal with the exception of notched P.

*Orthodiagram.* Normal size and shape of heart. Aortic loop rather full-sized.

*Follow-up, 7 yrs later.* Only considerable effort would bring on dyspnea. Findings: The heart was enlarged, there was noted a well-marked apical systolic murmur transmitted to the surroundings, and a systolic murmur of minor degree over the base.  $A_2$  was accentuated. There was no gallop nor evidence for failure. B.P.: Systolic from 130 to 190, diastolic from 78 to 110. The laboratory findings were essentially normal except for albuminuria. ECG: As compared with the previous study, the following changes were noted: The QRS complex showed an increase in voltage, and there was  $lt$  axis dev., the S-T interval was depressed in lead I,  $T_1$  was negative, and  $T_2$  positive.

*Orthodiagram.* The heart is enlarged, chiefly in the  $lt$  ventricular portion. A marked degree of tortuosity of the aorta is noted.

*Comment.* This patient was afflicted by a fluctuating type of hypertension, and developed heart murmurs of atherosclerotic origin. Orthodiagrams and ECG's obtained seven years apart, at different points in the course of the disease, illustrate the development of  $lt$  ventricular enlargement. Marked tortuosity of the aorta developed during this period.

ease is present. In those instances where the basal cardiac work (as determined by the ethyl iodide method) is not increased, the cardiac shadow is not found enlarged; the amount of enlargement is stated to be closely proportional to increased basal cardiac work. Some deepening of the waist of the silhouette (an aortic configuration) is common. An exception is found in individuals who represent, to begin with, a vertical type of the cardiovascular shadow; particularly with the drop heart the changes may be minimal and detectable only by means of follow-up studies conducted over a period of years. Other factors which will counteract the trend to progression in heart size are weight reduction and acceptance of the necessity for a quiet life. To some patients this latter require-



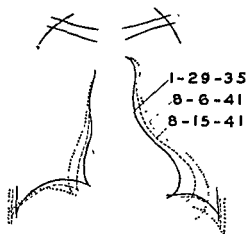


FIG 92—47 and 53½ yrs, M. History. When first seen the patient had had the anginal syndrome for one month Findings (age 47): Wt 84.1 kg (185 lb) There was no evidence of failure A slight degree of lt. ventricular hypertrophy and accentuation of  $A_2$  was noted Rate 84/min, rhythm regular BP 170/100, ECG Evidence of anterior infarction, no axis dev Other laboratory findings were without significance

*Orthodiagram* (1-29-35). The lt ventricular area is slightly enlarged and the oblique diameter measures 14 cm The lt atrium is not enlarged The lung fields showed normal transparency

*Course:* Three yrs later a minor posterior infarction occurred Two mos

later a minor anterior infarction, the patient making a nice recovery. Findings (3-29-38) showed no change in heart size (illustr omitted) nor in the blood pressure level Anginal distress was very slight Three and one-half yrs later insomnia developed A few wks later acute pulmonary congestion occurred during sexual intercourse In spite of this experience and weakness, the patient did not report until 10 days later Findings (age 53½) Wt 83.2 kg (183 lb) Cheyne-Stokes respiration, a few rt sided basal rales were noted, the liver was questionably enlarged, and the neck veins showed a normal systolic collapse The lt ventricle was much enlarged and hypertrophied, a gallop and basal accentuation were heard, there were no murmurs Rate 100/min, rhythm regular BP 180/95 ECG Similar to previous tracings except that lt. axis dev was present

*Orthodiagram* (8-6-41) The cardiac silhouette is much enlarged, with particular reference to the lt ventricular area, and the oblique diameter measures 19.8 cm The lt. atrium participates in the enlargement The pulsations were very small The lung fields showed a marked diminution of transparency, the vascular markings were increased and hazily outlined but there was no basal fluid

*Course:* Treatment consisted in bedrest, opiates for several nights, digitalis, aminophyllin by rectum, limitation of food, fluid and salt. 5 days later improvement was outspoken Findings The pulmonary rales and gallop had disappeared Auralicular fibrillation was noted and verified electrocardiographically, there was no pulse deficit, and the ventricular rate was 72/min BP 180/100 3 days later improvement had progressed Findings: Wt 80 kg (176 lb) Sinus rhythm present, rate 98/min BP 160/90

*Orthodiagram* (8-15-41) The size of the cardiac silhouette has markedly receded and the oblique diameter measures 16.1 cm A trace of lt atrial enlargement is noted The pulsations were fairly well visualized The transparency of the lung fields had increased and the vascular pattern was less marked

*Course:* Three weeks later sudden chest pain developed and death occurred within a few min

*Comment.* The patient had hypertension and obstructive coronary artery disease Angina of effort was present and several cardiac infarctions occurred 5½ yrs following the first observation acute left ventricular failure took place At that time the heart size had progressed to much enlargement Appropriate treatment soon led to clinical improvement, and simultaneously the heart had regressed in size That the regression in the size of the silhouette was not—or not chiefly—due to a resorption of pericardial fluid is made highly probable by the following facts. When the silhouette was largest, its vascular pedicle was not unduly shortened and fluid was not present in the pleural spaces. To judge from the increased thoracic width at that time, the volume of the abdominal viscera must have been increased, yet the diaphragm was depressed, obviously due to the weight of the blood in the excessively dilated cardiac cavities

ment is temperamentally acceptable, to others it may be re-enforced by the added imperative of other illness, but medical advice should emphasize the need for its acceptance.

As far as prognosis is concerned, the size of the heart is of limited significance. Large hearts may compensate for a period of many years, while smaller ones may fail quickly.

A correlation study has been carried out showing the comparative relation of roentgenologic and ophthalmoscopic findings and of roentgenologic and electrocardiographic findings. Heart size was evaluated from fluoroscopic and orthodiagraphic studies, the ophthalmoscopic observations referred to the degree of arteriolosclerosis of the hypertensive type, and the E C G. alterations were scrutinized with regard to final deflection changes and axis deviation. It was demonstrated that although there is a trend toward a positive correlation between roentgenologic findings as to heart size and the respective findings of ophthalmoscopy and electrocardiography, respectively, the degree of correlation is not of high statistical significance. The same held true when the correlation was carried out for electrocardiographic as compared to ophthalmoscopic findings. Although there is an inadequate correlation between these three criteria, it is desirable in a given case of hypertension to have an evaluation of heart size, eye ground and electrocardiogram.

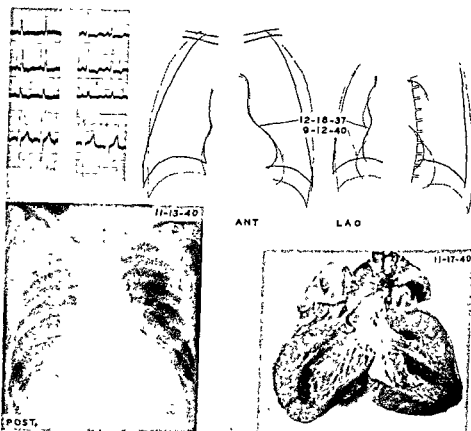
An increase in the size of the heart was observed in dogs in which hypertension had been produced by bilateral constriction of the renal arteries. None of the animals developed signs or symptoms of congestive heart failure.

Congestive failure is practically always associated with a considerable enlargement of the silhouette. Angina pectoris, however, is compatible with normal cardiac size. Many attacks of angina pectoris are associated with a paroxysmal hypertensive state. It would be interesting to study the size of the aorta during an attack and in the intervals between attacks. With progressive enlargement, the waist of the heart tends to straighten out, there is an in-

crease in the width of the heart shadow. This may well be that of an organic mitral valvular disease. But in contradistinction to a primary mitral heart, the aortic shadow, especially in the region of the knob, remains clearly visible, while with the increase in height of the mass of the heart shadow, and with the presence of right sided failure, a previously convexly-outlined right vascular contour may be replaced by the straight contour of the enlarged superior vena cava. At this stage pulsations have diminished markedly and the lung fields show signs of congestive lung failure.

**SUDDEN HYPOTENSION.** This occurs with a disturbance either of the central nervous system or of the heart. It is taken along with the patient

bral anemia are allied with it. Only a few roentgenological observations exist. Occasionally one is made during fluoroscopy when the patient begins to faint.



FIGS 93, 94, 95—43 and 46 yrs, M. History. Nocturia was present since childhood. The patient had developed ascites. Not until then had dyspnea of effort and digestive disturbances appeared. Several paracenteses had been done, an elevation of the B.P. was found. Findings. There was no dyspnea, no cyanosis, no engorgement of the neck veins. There was present some pretibial edema, ascites, and enlargement of the liver. Nothing particular was noted with respect to the chest wall and the heart except an accentuation of A, B.P. 180/115. The blood showed hb. 95 gms, rbc 4.3 mill, 58 gms protein with normal a/g ratio, Van den Bergh was normal, urea n between 22 and 30 mgm%. Special tests showed marked impairment of kidney function. Fundi. Retinal arteriosclerosis, grade II plus, hypertensive type. ECG (no preceding digitalis medication) N.s.r., initial deflection 0.08 sec wide, slurred, no axis dev, S-T segment slightly depressed in limb leads, and T of low voltage in leads I, II.

*Orthodiagrams* (ant and lt ant obl views) The diaphragm is elevated in conjunction with the ascites. The heart is practically normal in size. While there is some overlapping with the spine, in the lt ant obl view, this could be attributed to the elevation of the heart. The ascending portion of the aorta is somewhat prominent. Fluoroscopy revealed the pulsations along the cardiac borders to be at the lower limit of normal, and those along the aortic contours at the upper limit, the lungfields showed normal transparency.

*Course.* Ascites and edema of legs disappeared. B.P. elevation persisted. Within one year exertional dyspnea appeared, and cardiac enlargement was noted clinically. Findings. Wt: 55.6 kg (122 lb). Respiration was deepened, there was no evidence for congestive heart failure. There were no signs of pulmonary congestion. There was no evidence of anemia, urea n was 37 mgm%, creatinine 0.8 mgm%, CO<sub>2</sub> combining power was 53 vol%. Fundi. Retinal arteriosclerosis, grade III plus, hypertensive type,

mild edema of discs and several fine retinal hemorrhages

**ECG** (no preceding digitalis medication): As compared with 3 yrs prior, the initial deflection again shows absence of lt axis deviation, but its voltage is somewhat greater in leads I, II, CF. In leads I and II the S-T segments are definitely depressed, and the T deflections negative

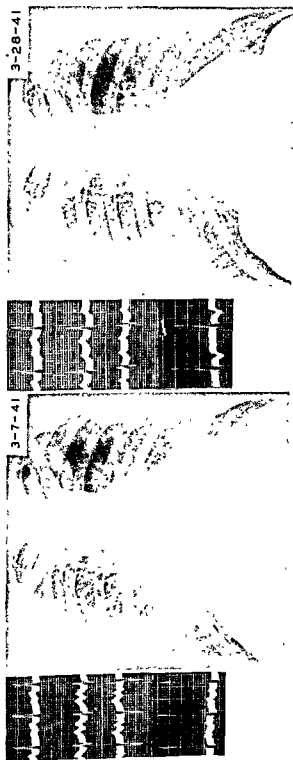
**Orthodiagrams** (ant. and lt. ant obl views) As compared with 3 yrs. prior, we note: Lung volume has obviously increased, the diaphragm is lower in position The heart has much increased in size, and this refers particularly to the lt ventricle Fluoroscopy revealed a moderate degree of pulmonary congestion and a diminished amplitude of diaphragmatic movements

**Course:** The patient was placed on digitalis He grew progressively worse. Two months later paroxysmal, nocturnal pulmonary congestion appeared Findings: A uremic picture was present with peripheral edema and pulmonary congestion but no fever The heart revealed a gallop rhythm, and soon afterward a pericardial friction rub appeared BP. 240/160 The urine showed 450 mgm % of albumin and a few casts The blood showed hb 7.5 gms, rbc 3 mill, wbc 15000 with 95% polys, the urea n was 120 mgm %, creatinin 8.2 mgm %, the plasma CO<sub>2</sub> combining power 36 vol %, protein and a/g ratio normal Fundi: In addition to retinal arteriosclerosis, there were noted angiospasm, hemorrhages, cotton wool exudates, edema of discs and retinae, and engorgement of retinal veins

**Post view:** The silhouette reveals an aortic configuration Basal fluid—more at lt than at rt—is noted In addition to vascular engorgement, the lung fields show also a mottled type of density Note The difference in density between the rt and lt half of the chest is due, at least in part, to extraneous technical conditions

**Postmortem** (4 days later) The weight of the heart, including the great vessels, was 680 gms. The apical portion of the heart was much rounded The rt ventricular cavity was slit-like in shape, its wall was 0.6 cm thick The lt. ventricular cavity was also narrowed, measuring 3.7 cm wide and 7.7 cm high, its wall was 2.2 cm thick, and the posterior papillary muscle measured 1.6 cm in diameter The valves appeared normal, and the coronary arteries showed non-obstructive atherosclerosis The aorta showed atherosclerosis, its diameter above the aortic cusps was 6.5 cm The heart muscle revealed no outspoken degenerative changes, neither macro- nor microscopically A uremic pericarditis was present Both pleural cavities contained fluid 600 cm<sup>3</sup> in the lt. and 100 cm<sup>3</sup> in the rt The lungs showed emphysema, mild congestion, suppurative bronchitis, and multiple, small and firm reddish areas that proved, on microscopic study, to be acute congestion with actual small hemorrhages There was no evidence for infarction. The liver showed only enlargement with congestion The kidneys were small and white, weighed 75 and 80 gms, the microscopic diagnosis was arteriolonephrosclerosis of malignant type superimposed upon a benign hypertension

**Comment:** A 3 yr follow-up study of a patient afflicted by benign hypertension, turning into the malignant phase with death resulting from renal failure Several features are of particular interest a) Clinical, when studied for the first time The presence of ascites and edema of the lower extremities was a diagnostic problem Heart failure did not account for it, portal cirrhosis and—as a remote possibility—constriction about the inf vena cava were thought of, but could be ruled out by the further course and on the basis of the postmortem study Nutritional-anemic factors may have accounted for it Lt ventricular hypertrophy certainly must have been present, yet no clinical evidence could be elicited This is to be accounted for by the absence of any enlargement b) Electrocardiographic The absence of lt axis deviation persisted in spite of progression of lt ventricular hypertrophy The cause for this is not clearly understood, for while emphysema was present yet the rt. ventricle remained small as compared with the lt ventricle As the QRS area increased in leads I and II, the S-T and T alterations in these leads became more marked c) Roentgenologic The ascending limb of the aorta was prominent, yet there was no enlargement found anatomically, the dilatation observed during life was, therefore, dynamic in nature Progression in the size of the lt. ventricle may be studied from both the ant and lt ant obl views. The mottled and patchy pulmonary changes were due to focal areas of congestion and hemorrhage Lt ventricular failure with normal rhythm favors fluid accumulation in the lt pleural cavity, this was observed in the roentgenogram and verified anatomically.



FIGS 96, 97—53 yrs, M History The patient, a physician, was afflicted by a throat lesion. He had been working hard up to the day of the examination. His fluid intake was considerable and included beer. He reported transient elevations of his B P, that there had been no anginal distress and no disturbing degree of effort dyspnea, that he had perspired easily for the past few mos and that he had been aware of his heart action for the past few days Findings Wt 75.5 kg (166 lb) The veins in the neck were somewhat distended but still revealed systolic collapse, the liver was not enlarged and there was no edema Bilateral basal rales were heard The left ventricle was enlarged, and the apical thrust was widened and heaving An outspoken gallop was noted,  $P_2$  was accentuated, and there was a low pitched systolic apical murmur transmitted to the lt The rate was 114/min, and the B P 180/130 The blood count was normal, Wassermann test negative, the serum calcium 10.1 mgm % but when repeated in another laboratory a few days later, 8.1 mgm % The urine showed 100 mgm % of albumin and 0.3 gm. of sugar in 100 cc Biopsy of the larynx revealed grade III of squamous cell carcinoma ECG:  $Sr$ ,  $P$  widened to 0.12 sec,  $P-R$  interval 0.20 sec, initial deflection with lt axis dev,  $T$  of low voltage in I,  $CF$ ,  $Q-T$  segment prolonged to 0.34 sec. (upper limit of normal for cycle length of 0.526 sec being 0.319 sec)

Ant view: The lung vessels are enlarged and hazily outlined, and the air content of the lung fields is diminished The pleural spaces are free of fluid The silhouette is considerably enlarged

The rate slows considerably and the silhouette widens in a globular fashion. Those with marked tachycardia will perhaps reveal a narrowing of the silhouette but observations along this line are needed. In two other observations a marked fall in the blood pressure was induced by moderate exertion, and the heart rate showed no marked increase; the aortic shadow was noted to decrease considerably, and the size of the aortic arch diminished as much as from 3.9 to 2.6 cm. in the one instance, and from 2.9 to 2.3 in the other.

**CHANGES IN FILLING.** With a marked loss of blood, the time comes when the amount of fluid returning to the heart is so limited that size and stroke volume must decrease. Secondary entrance of tissue fluids and stagnant blood maintain the circulation. The roentgenological heart size decreases in dogs when about 20% of the blood volume is removed by venesection, but in about one hour the silhouette has returned to its original size. The same amount of blood injected into a dog leads to a corresponding increase in the silhouette, but for a shorter period. This increase seems to be associated with a simultaneous increase in venous pressure. The effect of blood loss in man has been studied in blood donors with a loss of eight to ten percent of the total blood volume within a few minutes. The reported findings of a decrease in the area of only 2-6% found by correction from films taken at 100 cm. distance do not permit of any conclusions whatever. No definite results as to changes in size were found with the ordinary roentgen technique in cases where therapeutic venesection was undertaken because of heart failure, except that the degree of congestive lung failure diminished. Two cases with great enlargement, however, have been studied by means of the volumetric reconstruction method and volumetric differences were found, amounting to as much as 500 cm<sup>3</sup>.

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






*Course.* Bed rest, fluid limitation, digitalis, and opiates for laryngeal pain were advised. This was carried out elsewhere, and calcium and vitamins were added. Reexamination 18 days later. Findings: Wt 70.9 kg (156 lb). The patient felt better. The basal breath sounds were harsh but rales had disappeared. The area of cardiac dullness seemed diminished but the character of the apical sounds was unchanged. Gallop was not present, the systolic apical murmur was of low intensity. The rate was 84/min, and the BP 175/105. A glucose tolerance test showed a diabetic curve. Fundi: Grade I plus of retinal arteriosclerosis, hypertensive type. ECG: S-R with a few premature ventricular beats, P widened to 0.12 sec, P-R interval prolonged to 0.22 sec, lt axis dev, S-T and T reveal digitalis effect, Q-T segment 0.35 sec, normal (upper limit of normal for cycle length of 0.714 sec being 0.376 sec).

*Ant view.* The appearance of the lung vessels and lung fields is nearly normal. The silhouette is moderately enlarged to the lt. As compared with the previous study, the level of the diaphragm is caudad by about half of a dorsal interspace.








*Course.* The patient was kept in bed on a maintenance dose of digitalis, fluid restriction, and a diabetic diet. He received daily radiation treatment to the neck, a total of 4000 r units in the course of 4 weeks. After that period his wt was 65.9 kg (145 lb). The rate was 84/min, and the BP 160/90, and the apical murmur was nearly gone. 3 mos later the laryngeal lesion had disappeared. The cardiac findings were unchanged, there were no complaints, and part-time professional activity had been resumed. One year later local recurrence requiring tracheotomy.

*Comment.* The patient had developed left ventricular failure. This was accidentally discovered. Proper management led to improvement, which found its roentgenologic expression in regression of heart size and disappearance of pulmonary congestion.

## A

CARDIAC ENLARGEMENT	RETINAL CHANGES			
	I	II	III	
NONE	10	3	0	 77/23/0 %
MODERATE	14	14	2	 47/47/6 %
MARKED	9	5	2	 56/31/13 %
AVERAGE CARDIAC ENLARGEMENT				
	22 51 27 %	30 43 27 %	13 64 23 %	0 50 50 %
				 56/37/7 %
				AVERAGE RETINAL CHANGES

## B

ECG ALTERATIONS	CARDIAC ENLARGEMENT			
	NONE	MOD- ERATE	MARKED	
FDC ABSENT LAD ABSENT LAD PRESENT	6 5 1	11 6 5	3 2 1	 30/55/15 %
FDC PRESENT LAD PRESENT LAD ABSENT	7 5 2	19 16 3	13 11 2	 18/49/33 %
AVERAGE ECG ALTERATIONS				
	34 66 %	46 54 %	37 63 %	18 82 %
				 22/51/27 %
				AVERAGE CARDIAC ENLARGEMENT

rows has been totaled, and the percentage of cases with  
percentage has been plotted as a divided horizontal bar, so that, regardless of the number

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The following four case reports are interesting in this connection. One patient with cachexia due to undernourishment received twice an infusion of 850 cm<sup>3</sup>. of saline and at both times the T and A figures for the silhouette increased 11-12%.—Another patient was much dehydrated due to vomiting and diarrhea. The infusion of 1030 cm<sup>3</sup>. of saline increased both T and T<sub>D</sub> by 11%.—A man, a printer by profession, suffered from lead poisoning and vomited excessively. His body weight was 45.5 kg., the hb. was 72% and the r b c. 3.8 millions. The blood plasma volume was 27.5 cm<sup>3</sup>. and the blood volume 55 cm<sup>3</sup>. per kg. body weight T was 8.3 cm. The vomiting was treated by liver therapy. 20 days later the body weight was 50.4 kg., the hb. was 66% and the r b c. 3.84 millions.

of cases, the relative lengths of the bars indicate the distribution into the classes. The sums of these horizontal bars have been treated in the same way, giving an additional bar, indicative of the average distribution of these selected cases. Each of the vertical columns has been likewise totaled, and the percentage of each class has been computed. This percentage has been plotted as a divided vertical bar, so that, regardless of the number of cases, the relative lengths of the bars indicate the distribution into the classes. The sums of these vertical bars have been treated in the same way, giving an additional vertical bar, indicative of the average distribution of these selected cases.

An analysis of table A reveals. The horizontal bars indicate an increase of group III retinal arteriosclerosis with progressive change in heart size. The progression for group II is not regular. However, if one fuses the rows for moderate and marked cardiac enlargement, the increase for group II is quite striking, as compared to the average (lower right hand corner), and as compared to the percentage of cases with normal heart size. The vertical bars indicate in general an increase in cardiac enlargement accompanying progression in retinal vascular sclerosis. Certainly, as retinal pathologic changes increase, the incidence of no cardiac enlargement is progressively reduced, and in this series normal heart size does not happen to coincide with group III retinal arteriosclerosis (third row, first column). Exceptions to this positive correlation are shown in those hypertensive patients who have an association of normal radiological heart size with a higher degree (group II) of retinal arteriosclerosis (three cases, first row, second column). Conclusion: There is a trend, towards a positive correlation between cardiac enlargement and the grade of the retinal arteriolar changes.

An analysis of table B reveals. The horizontal bars indicate an increase of cardiac enlargement in cases with electrocardiographic final deflection changes, as compared to the average (lower right hand corner), and as compared to the cases with absence of final deflection changes. The vertical bars indicate a steady increase in the percentage of cases with electrocardiographic final deflection changes as the radiologic examination shows an increase in the size of the heart. It is possible for hypertensive patients to show an association of final deflection changes, without left axis deviation, with a normal heart size (two cases, first row, second column). Conversely, one finds that an absence of final deflection changes and of left axis deviation, is compatible with a marked degree of cardiac enlargement (two cases, third row, first column). Conclusion. There is a trend towards a positive correlation between electrocardiographic changes and cardiac enlargement.

It has thus been demonstrated that with essential hypertension there is a trend towards a positive correlation between roentgenologic findings as to heart size and ophthalmoscopic and electrocardiographic findings, respectively, but that the degree of correlation is not of high statistical significance. The same holds true if the correlation is carried out between electrocardiographic and ophthalmoscopic findings. Since there is an inadequate correlation between the three criteria in question, it is desirable to have, in a given case of essential hypertension, an evaluation of heart size, eyeground and electrocardiogram. From Roesler, H., Gibson, G. G., Hussey, R., *Ann Int Med*, 1940, 13, 1814. Courtesy *Ann Int Med* and Lancaster Press, Lancaster, Pa., publisher.



The blood plasma volume was 53 cm<sup>3</sup>. and the blood volume 82 cm<sup>3</sup>. per kg. body weight. T had risen to 10.6 cm—A very small cardiac silhouette was noted in a young man who had suffered a severe intestinal hemorrhage. The body weight was 50.5 kg. and the hb. 24%. Four months later the weight had risen to 72.5 kg. and the hb. to 87%. T had increased 13% and A 19%, with the position of the diaphragm nearly identical.

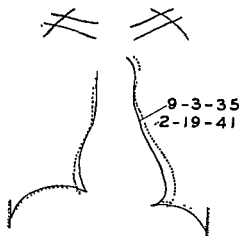


FIG. 99—21, 24 and 30 yrs, M History. In school up to the age of 18 the patient had been active in swimming, tennis and track running. He then became introspective, over-cautious, and discontinued all physical activity. Complaints referred to tiredness, also an elevation of the B.P. had been noted. Findings (age 21). Ht. 185 cm (6 ft 2 in), wt 70.5 kg (155 lb.). Introvert personality showing marked vasolability. The heart was negative except for a rate of 120/min. BP 155/85. The ECG showed a tendency to rt ax dev. Fluoroscopy showed for the upright position a rather small heart shadow in vertical position and, with tachycardiac action, for the recumbent position the size of the heart definitely increased and the rate slowed considerably.

*Course* The patient gained some self-confidence and gradually improved. He still exercised but little. Findings (age 24). Wt 72.4 kg (159 lb.). The cardiac findings were normal, the rate was 106/min and the B.P. 145/80. ECG: Unchanged.

*Orthodiagram:* Vertical silhouette of nearly normal size.

*Course* The patient was advised to be physically more active. He worked now hard for 2 yrs assembling heavy engines, did calisthenics, walked 2 miles every evening, skated, etc. Findings (age 30). Wt 76.4 kg (168 lb.). The cardiac findings were normal, the rate was 83/min and the B.P. 125/80. ECG: The degree of rt ax dev. lessened, T<sub>1</sub> were of better voltage.

*Orthodiagram* As compared with the findings of 5½ yrs ago, an increase in the size of the silhouette is observed, also the aortic knob is more conspicuous. There is a slight increase in the width of the chest.

*Comment* The increase in heart size here observed is attributed to a better filling in connection with the beneficial effect of prolonged physical activity on the body tonus. Increase in age, of course, cannot account for the change in heart size and probably does not account for the change in the size of the aorta, which is also better filled. Increase in weight does not account for an apparent increase in heart size due to change in position because very little alteration in the height of the diaphragm is noted.

Certain small hearts are found to be small only when examined in the vertical position. Owing to deficient muscular tonus of the diaphragmatic, abdominal and peripheral muscles, possibly also by reason of vasomotor weakness, blood fails to enter the general circulation but returns if the subject assumes a horizontal position and the silhouette immediately increases. It also increases when the patient is examined while standing in water up to the level of the umbilicus. It may be found to be increased during an examination in the upright position when elastic bandages have previously

been applied in the recumbent position. Such observations have been made on a variety of patients, all tending to suffer from dizziness, headache, palpitation, psychic and physical exhaustion, and showing a tendency to low blood pressure levels and to tachycardia, and all improving in the recumbent position. Some are of the asthenic-gracile type, some are or have been recently afflicted by infectious-toxic conditions, in others there are present diseases of the central nervous system that have been referred to previously. Since the factors regulating the blood flow to the heart vary considerably at different times and even within a short period, the degree of orthostatic diminution may vary considerably for a given patient. Thus it seems justified to state that the influence of the filling of the heart on its size is underrated and that the constitutional factor is often too greatly stressed. One should make a diagnosis of constitutionally small and hypoplastic heart only when the heart is found to be small under conditions of optimal filling, i e., in the recumbent position, and when it is not accounted for by either peripheral vasomotor failure or a diminution of blood volume, such as may, for instance, accompany diarrhea or cachexia.

Suprarenal insufficiency deserves special consideration. The diminution in cardiac volume is very outspoken when a crisis is impending in Addison's disease. This coincides with a loss of sodium and water and with a low blood pressure. A normal heart size is regained following adequate treatment with cortical extract. An overdosage of the hormone, particularly when associated with an injudicious salt and water intake, may bring about cardiac enlargement and congestive failure. Patients in the critical phase of Addison's disease die readily on effort and therefore roentgenologic studies are better avoided or at least limited to the recumbent position.

The problem of posture and circulation was approached in the following instructive experiment. Rabbits have a poorly developed mechanism to counteract the effect of gravity on the circulation. When kept in the upright position, circulatory and respiratory failure results; the collapse time is 30 to 160 minutes for male and 10 to 25 minutes for female animals. A roentgenogram taken toward the end of the collapse shows a narrowing of the cardiac silhouette, and postmortem study reveals the cardiac cavities empty of blood. Cutting the aortic depressor and carotid sinus nerves results in vasoconstriction, the collapse time is markedly prolonged, the diminution of the size of the silhouette delayed, and postmortem study reveals the cardiac cavities partly filled with blood; the right side more so than the left.

In man, a ligature about both upper femoral regions may decrease the size of the silhouette, as does also pressure at the site of an arteriovenous anastomosis.

**ANEMIA** With a severe degree of anemia—the hemoglobin falling below 50%—the work of the heart has to increase appreciably. If such an anemia lasts for a long period, some degree of cardiac enlargement may result. It does not seem to matter whether the anemia be of the primary macrocytic type or of the secondary type due to hemorrhages or to parasitic infections such as *Botriocephalus*, *Ankylostoma* or *Plasmodium malariae*. A tendency to enlargement will be fostered by considerable demands on the circulation, such as occasioned by heavy work, pregnancy or fever. Anemia of nutritional origin may

be associated with Thiamin deficiency. Numerous occlusions of the small pulmonary arteries have been associated with the cardiac enlargement noted in sickle cell anemia. Functional cardiac murmurs, occasionally even diastolic in character, are commonly present. An exaggerated type of activity is observed fluoroscopically, possibly in connection with the increase in blood velocity. With a marked improvement of the blood picture, this hyperactivity is noted to disappear. The presence of haziness of the lung fields together with denser, wider, hilus shadows indicates circulatory insufficiency, as also do the findings of arterial oxygen deficiency, venous hypertension and cardiac dilatation. These lung findings are reversible in direct proportion to the improvement of the blood picture. Convincing observations have been made of the regression in heart size which accompanies an improvement in the blood picture. In other instances the roentgenologic technic used was not sufficiently exacting to permit accurate observations on the comparatively small changes in cardiac dimensions. A comparison of the heart size becomes impossible in those cases of anemia which are connected with leukemia, in which the size of the spleen undergoes changes during the course of therapy. A report on 3 members of a family who were affected by hemolytic icterus with associated cardiac enlargement is interesting; following therapeutic splenectomy the silhouette became normal in size in each case.

**POLYCYTHEMIA.** The total blood volume is considerably increased in this condition. Anatomical studies have revealed that the pulmonary artery and its branches are considerably dilated; usually more than the vessels of the greater circulation. Vasomotor influences seem to play a minor rôle for the lesser circulation so that the amount of blood has a determining influence on the size of the vessels. The vascular shadows appear markedly increased in the roentgenogram, often with an additional areolar pattern and fine stippling similar to the appearance in miliary tuberculosis and certain cases of mitral

flowing  
stance,  
by phenylhydrazine), the transparency of the lung fields was observed to increase, and there was an approach of the vascular pattern toward a normal appearance.

**STARVATION.** The heart diminishes in volume and in weight. This has been shown to be most marked in children and adolescents. Dogs were submitted to inanition until they lost about one-third of their body weight. The roentgenological determination of the area in two directions revealed a diminution which varied from 8 to 23%. In the dog which showed the 23% diminution, the actual loss in blood volume was determined to be 51%. The decrease in heart size takes place rather slowly, but during a subsequent feeding period the former size is rapidly attained.

**Physical Agents and Poisoning.** The problem of diminished air pressure is of interest in the fields of aviation and mountain climbing. Two factors interplay; the low air pressure and low partial oxygen pressure. Percussion in a low air pressure chamber, corresponding to an altitude of 5-7000 m., showed dilatation. It was likewise found in those members of the Himalaya expedition who nearly gained the summit, and it was thought to take from seven to

fourteen days for a complete return to normal. In no instance was dilatation found roentgenologically in the members of the Andes expedition (at about 4700 m.). Attempts to study the heart size roentgenologically in a low air pressure chamber met with great difficulties. The respiration became rapid and deep. The expansion of the intestinal gas, the volume of which doubles at an altitude of 6000 m., drives the diaphragm cranially. The individuals who submitted to the experiment became restless, tired, uncoöperative, underwent convulsions, and had to be forcibly restrained from making violent movements. Where parts of the roentgen equipment (tube and high voltage wires) are enclosed in the chamber, special insulation is necessary to prevent sparks at such low pressures, and in addition the output of energy falls considerably. This problem remains to be studied along with the study of the heart size when only the oxygen content is lowered. It is known that respiration of air which contains only 8% of oxygen often causes electrocardiographic changes and sometimes cardiac pain, both thought to be an expression of anoxemia of the heart muscle.

When the breath is retained sufficiently long, asphyxia sets in and the silhouette, by fluoroscopic examination, is seen to broaden after 50 to 60 seconds have elapsed. When respiration with 30% carbon dioxide is induced in anesthetized dogs a considerable widening of the silhouette develops which persists for many hours. The hearts of these sacrificed animals show a marked dilatation, especially of the right ventricle, and poor tonus of the muscle.

Poisoning with *illuminating gas* may be added here. The blood pressure falls considerably and the postmortem examination reveals necrosis and hemorrhage of the heart muscle and enormous vascular dilatation. Dilatation of the heart has been reported roentgenologically but does not seem to be a constant finding. The hearts of rabbits dilate, although cats demonstrate no changes. Cardiac dilatation with rapid changes has been reported for intoxications with war gases (oxychloride, surpalite). Cardiac enlargement accompanied by increased pulsations along the pulmonary artery has been noted in some patients afflicted by so-called endemic dropsy. This syndrome is observed in India and is probably caused by the ingestion of mustard oil that has been adulterated with oil obtained from *Argemone mexicana*.

An increase in the external temperature tends to increase the circulating blood volume. The size of the cardiac silhouette was determined orthodiagraphically following hot and cold baths and it was found to be usually diminished after the former and usually increased after the latter. Since the diaphragm was not always found to be identical these experiments should be repeated employing a careful roentgenographic technique.

Operation. Roentgenologic studies of heart size and activity during the stage of operation have not been reported. Postoperative findings do not necessarily permit us to draw conclusions as to the operative period. Certainly uniform changes cannot be expected when one considers the many influencing factors such as the status of the cardiovascular system, the type of anesthesia, blood loss, changes in blood pressure, degree of anoxemia and diminished activity of the respiratory muscles. Possible differences in the diaphragmatic position interfere with a correct comparison of roentgenological records. Two authorities have compared the pre- and post-operative orthodiagraphical find-

be associated with Thiamin deficiency. Numerous occlusions of the small pulmonary arteries have been associated with the cardiac enlargement noted in sickle cell anemia. Functional cardiac murmurs, occasionally even diastolic in character, are commonly present. An exaggerated type of activity is observed fluoroscopically, possibly in connection with the increase in blood velocity. With a marked improvement of the blood picture, this hyperactivity is noted to disappear. The presence of haziness of the lung fields together with denser, wider, hilus shadows indicates circulatory insufficiency, as also do the findings of arterial oxygen deficiency, venous hypertension and cardiac dilatation. These lung findings are reversible in direct proportion to the improvement of the blood picture. Convincing observations have been made of the regression in heart size which accompanies an improvement in the blood picture. In other instances the roentgenologic technic used was not sufficiently exacting to permit accurate observations on the comparatively small changes in cardiac dimensions. A comparison of the heart size becomes impossible in those cases of anemia which are connected with leukemia, in which the size of the spleen undergoes changes during the course of therapy. A report on 3 members of a family who were affected by hemolytic icterus with associated cardiac enlargement is interesting; following therapeutic splenectomy the silhouette became normal in size in each case.

**POLYCYTHEMIA** The total blood volume is considerably increased in this condition. Anatomical studies have revealed that the pulmonary artery and its branches are considerably dilated; usually more than the vessels of the greater circulation. Vasomotor influences seem to play a minor rôle for the lesser circulation so that the amount of blood has a determining influence on the size of the vessels. The vascular shadows appear markedly increased in the roentgenogram, often with an additional areolar pattern and fine stippling. The appearance in miliary tuberculosis and certain cases of mitral regurgitation, have been observed.

Following therapeutic reduction of the red blood cells and blood volume (for instance, by phenylhydrazine), the transparency of the lung fields was observed to increase, and there was an approach of the vascular pattern toward a normal appearance.

**STARVATION.** The heart diminishes in volume and in weight. This has been shown to be most marked in children and adolescents. Dogs were submitted

actual loss in blood volume was determined to be 51%. The decrease in size takes place rather slowly, but during a subsequent feeding period the former size is rapidly attained.

**Physical Agents and Poisoning.** The problem of diminished air pressure is of interest in the fields of aviation and mountain climbing. Two factors interplay; the low air pressure and low partial oxygen pressure. Percussion in a low air pressure chamber, corresponding to an altitude of 5-7000 m., showed dilatation. It was likewise found in those members of the Himalaya expedition who nearly gained the summit, and it was thought to take from seven to

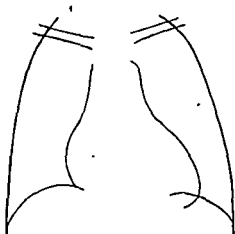
fourteen days for a complete return to normal. In no instance was dilatation found roentgenologically in the members of the Andes expedition (at about 4700 m). Attempts to study the heart size roentgenologically in a low air pressure chamber met with great difficulties. The respiration became rapid and deep. The expansion of the intestinal gas, the volume of which doubles at an altitude of 6000 m., drives the diaphragm cranially. The individuals who submitted to the experiment became restless, tired, uncooperative, underwent convulsions, and had to be forcibly restrained from making violent movements. Where parts of the roentgen equipment (tube and high voltage wires) are enclosed in the chamber, special insulation is necessary to prevent sparks at such low pressures, and in addition the output of energy falls considerably. This problem remains to be studied along with the study of the heart size when only the oxygen content is lowered. It is known that respiration of air which contains only 8% of oxygen often causes electrocardiographic changes and sometimes cardiac pain, both thought to be an expression of anoxemia of the heart muscle.

When the breath is retained sufficiently long, asphyxia sets in and the silhouette, by fluoroscopic examination, is seen to broaden after 50 to 60 seconds have elapsed. When respiration with 30% carbon dioxide is induced in anesthetized dogs a considerable widening of the silhouette develops which persists for many hours. The hearts of these sacrificed animals show a marked dilatation, especially of the right ventricle, and poor tonus of the muscle.

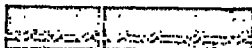
Poisoning with *illuminating gas* may be added here. The blood pressure falls considerably and the postmortem examination reveals necrosis and hemorrhage of the heart muscle and enormous vascular dilatation. Dilatation of the heart has been reported roentgenologically but does not seem to be a constant finding. The hearts of rabbits dilate, although cats demonstrate no changes. Cardiac dilatation with rapid changes has been reported for intoxications with war gases (oxychloride, surpalite). Cardiac enlargement accompanied by increased pulsations along the pulmonary artery has been noted in some patients afflicted by so-called endemic dropsy. This syndrome is observed in India and is probably caused by the ingestion of mustard oil that has been adulterated with oil obtained from *Argemone mexicana*.

An increase in the external temperature tends to increase the circulating blood volume. The size of the cardiac silhouette was determined orthodiagraphically following hot and cold baths and it was found to be usually diminished after the former and usually increased after the latter. Since the diaphragm was not always found to be identical these experiments should be repeated employing a careful roentgenographic technique.

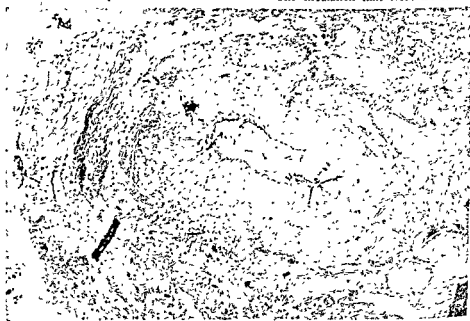
**Operation.** Roentgenologic studies of heart size and activity during the stage of operation have not been reported. Postoperative findings do not necessarily permit us to draw conclusions as to the operative period. Certainly uniform changes cannot be expected when one considers the many influencing factors such as the status of the cardiovascular system, the type of anesthesia, blood loss, changes in blood pressure, degree of anoxemia and diminished activity of the respiratory muscles. Possible differences in the diaphragmatic position interfere with a correct comparison of roentgenological records. Two authorities have compared the pre- and post-operative orthodiagraphical find-



COMPRESSION OF RT FEMORAL ART



FIGS. 100, 101—14 yrs, M History. Enlargement of the rt. leg had been present since birth Findings: Ht 125 cm (4 ft 11 in), wt 38.2 kg (84 lb). The rt. leg was enlarged, showed increased warmth, prominent veins, ulceration over the internal malleolus, and a systolic murmur and thrill over the femoral artery. The venous pulsations in the neck were increased and showed a normal systolic collapse. The heart was moderately enlarged, the apical thrust was heaving. A third sound (not gallop) and short systolic murmur were heard at the apex but disappeared on compression of the rt. femoral artery; furthermore, such compression changed the heart rate from 88 to 66/min and the brachial B.P. from 100/48 to 100/66. Oscillometric readings showed the amplitude at the rt. leg five times larger than at the lt. The circulation time from the femoral



veins to the head was a rt. leg vein at the saturation), which is (94.2% of saturation) in arterial blood. ECG: Normal findings

*Orthodiagram:* Silhouette moderately enlarged. Diameters. Oblique 128 cm, broad 93 cm, depth 94 cm, computed volume 504 cm<sup>3</sup>. Fluoroscopy showed an increased amplitude of pulsations. Compression of the rt. inguinal area did not cause a change in heart size but there was a suggestive diminution of the pulsations.

*Course:* Veins of the rt. leg were sclerosed or excised at different occasions. Micro-

ings; one found no uniform changes, while the other found an increase in the size of the area of the cardiac silhouette of from 8-12 cm<sup>2</sup>. and a diminution of the amplitude of pulsations only, in those cases where tests had revealed functional alterations of the myocardium. For further data the reader is referred to a discussion of disturbed function of the diaphragm (see chapter VI).

**Arterio-Venous Anastomosis.** Such an abnormal communication leads to a shunt of arterial blood into the venous system without passing through a capillary bed. Whether the anastomosis be acquired, traumatic or congenital, and whether the seat be in the greater circulation or, as rarely happens, in the lesser circulation, cardiac dilatation and hypertrophy result, provided the fistula is wide enough. Signs and symptoms of failure in both the greater and lesser circulation may develop. The stroke and minute volume is considerably increased as is also the heart rate and the pulse pressure. The cardiac silhouette in most cases shows a globular enlargement which is sometimes very considerable, there is often an increased amplitude and some prominence of the pulmonic arch; engorgement in the lesser circulation can be quite marked. Shutting off the shunt by simple compression may lead to a narrowing of the cardiac silhouette notwithstanding the resulting slowing of the heart rate. The narrowing may be limited to the right side or affect the entire silhouette. At the same time the extent of pulsation and prominence of the pulmonic arch may diminish together with the roentgenologic signs of congestive lung failure. Murmurs which have been present can disappear completely. When by surgical intervention the shunt is closed, a regression of the cardiac silhouette and of the abnormal lung pattern occurs within a comparatively short time along with a return to normal clinical findings.

Two other forms of arterio-venous shunts may be mentioned here. One is the interatrial septal defect which represents an intracardiac fistula. The other is represented by thyrotoxicosis and beriberi. Here a widening of the small arterioles permits a leaking of blood, without much resistance, from the end of the arterial into the capillary and venous systems.

**Deformity of the Chest Wall.** Typical anatomic changes in the shape and position of the heart and great vessels are found in connection with certain thoracic deformities. The knowledge of these thoracic cage deformities prevents the embarrassment of erroneous interpretation where a normal cardiovascular system actually is present. This is all the more important since the clinician finds it difficult to interpret his auscultatory and percussory findings in the presence of thoracic deformities and needs to rely much more upon the roentgenologic examination.

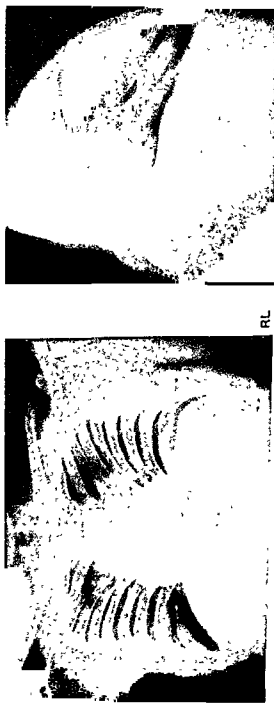
**KYPHOSCOLIOSIS** a) Right convex. A moderate degree of this deformity is rather common. The right chest cavity is anatomically diminished in all its dimensions while the left side, though diminished in height, has a greater

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scopic: The veins were thick-walled, suggesting that these vessels had taken over some of the functions of arteries (*microphotogram*). The cardiac findings remained unaltered.

*Comment:* An instance of congenital phlebarteriectasis of one extremity with secondary cardiac involvement.





FIGS 102, 103—33 yrs, M Tuberculosis of spine in childhood. Marked kyphosis and slight scoliosis. Dyspnea on effort and palpitation. Height 132 cm, weight 30 kg. Very slight cyanosis. Normal heart sounds. *Ant view*: height of chest and cardiac silhouette diminished. T 9 8, Th 18 7 cm. *Lat view*: the main axis of heart inclines on angle of approximately 25° with horizontal line. Depth of heart considerable. The heart size would be underrated by limiting the examination to the anterior view only.

volume. The anterior view shows the upper mediastinal shadow much enlarged. During the early days of roentgenology, a diagnosis of aneurysm or tumor was often made in these cases. A horizontal shift of the fluoroscopic tube, however, informs the examiner that this shadow moves much more than the cardiovascular shadow and is therefore located in a more posterior plane, i e., nearer to the target of the tube. This fact is likewise revealed by rotation of the patient, and also by means of a small shutter that makes possible better visualization of the transparent areas of the intervertebral discs. The mass of the cardiac silhouette is definitely too far to the left, and the left lower contour may almost reach the left inner chest wall contour in projection. The whole left contour is straight, the pulmonic arch prominent, the aortic knob is well seen and the aortic arch approaches the jugular notch. The aortic bed of the esophagus is flat and long. Further caudad there is often a slight indentation where the esophagus crosses the left main bronchus, but its whole course is only slightly curved and does not follow closely the course of the spine. The right anterior oblique view shows a deep retrocardiac space, and the shadow of the descending aorta is very clearly seen.

The straight left contour and the decreased distance to the left are changed into a silhouette of normal appearance as soon as the patient is rotated into the left anterior oblique view until the spine projects as a straight band. This procedure proves what can also be verified by a postmortem study, that the heart, in the presence of a right convex kyphoscoliosis has undergone a counter-clockwise rotation. When the kyphosis attains greater degree, the straight left contour becomes less pronounced.

b) Left convex. This deformity is less common than the former and is easily overlooked. Since the greater part of the cardiovascular silhouette is situated to the left, the  
into the left lung field.  
tion in the anterior view.

With the seat of scoliosis at the upper or middle third of the chest the vascular shadow appears much enlarged; and with a very pronounced degree of scoliosis a vertical transparent area is visualized in the center of this upper mediastinal shadow. With the maximum of scoliosis at a lower level, the median position persists but the vascular shadow is not broad. The barium-filled esophagus reveals a moderate deviation to the left without, however, closely following the shadow of the spine. The apparent aortic dilatation is obviously caused by the displacement of the shadow of the descending aorta combined with the deviation of the spine to the left. The transparent area is nothing else than the window of the aorta which is normally seen only after rotation into the left anterior oblique view. The descending aorta will also be displaced by a lower scoliosis but its shadow will be hidden behind the cardiac shadow, and its course is not interfered with at the level of the upper mediastinum. The normal size of the aorta is revealed in the oblique views.

The whole silhouette will appear normal if the patient is rotated into the right anterior oblique view until the spine projects as a straight band. This procedure proves that the heart has undergone a clockwise rotation in the presence of a left convex scoliosis, and that in addition the descending aorta is displaced.

**PURE KYPHOSIS** This is rather rare as an isolated deformity and is often

complicated by a slight degree of lateral curvature. The etiology is most commonly a tuberculous destruction of several vertebral bodies with a resulting angular kink of the spine. Unless the site of the angulation is at the uppermost level of the thoracic spine, the height of the chest is considerably diminished. The narrow intercostal spaces produce a diminution in the transparency of the lung fields, but the presence of a normal vascular pattern should prevent one from making the erroneous diagnosis of congestive lung failure. The cardiac silhouette, as observed in the anterior view, is in median position, relatively small, globular and dips into the abdominal shadow. The vascular shadow is also diminished in height, and the right contour is straight (unless marked aortic dilatation is present). The lateral view reveals a very deep chest, and the cardiac silhouette has the appearance of an egg lying on its



FIGS. 104, 105.—28 yrs. M. A funnel chest was acquired following burial under debris. No complaints. Physically well. Depth of funnel  $4\frac{1}{2}$  cm. Moderate tachycardia, otherwise clinical findings negative. Ant view silhouette in toto too far to fit. A portion of thoracic spine is visible above diaphragm. Because of displacement of heart, vascular structures of medial portions of rt lung have become clearly visible. Lt lat view depression of sternum (bl arrow) and ventral border of heart (wh arrow).

side as it rests broadly upon the diaphragm which ascends dorsad. The depth of this silhouette exceeds the transverse dimension in the anterior view and the normal ratio of  $T_D/T$  has changed from an average of about 73% to a maximum of 150%. The esophagus appears short in the anterior view and rather straight in its course. In the lateral view it is seen to deviate at the level of the diaphragm and to pursue a ventral course toward its diaphragmatic passage. Anatomic studies indicate the presence of a muscular contraction rather than an actual decrease in length. This lateral view proves that the heart, in the presence of a kyphosis, has undergone a displacement with lowering of the base as the outstanding feature. Its size is considerably under-estimated when the examination is limited to the anterior view.

**FUNNEL CHEST.** This deformity may be either congenital or acquired. The depression of the sternum is commonly found in its lower third. Anatomical sections demonstrate a displacement of the mass of the heart toward the left chest cavity and a marked diminution of the depth of the heart at the

level of the depression. The anterior view shows no striking changes in the silhouette which, on the whole, lies too far to the left; the right border may completely disappear within the spinal shadow. The density of the silhouette is somewhat diminished and the shadow of the spine is rather well seen through the right part of the silhouette. The esophagus deviates to the left of the midline. The lateral view reveals the depressed sternum in profile and the diminished antero-posterior dimension of the heart which may be only 50% of the transverse dimension as measured in the anterior view. High degrees of funnel chest may be associated with circulatory embarrassment, and operative procedures may lead to marked improvement and a return of the silhouette to its normal position.

In summarizing, it may be stated: Right convex kyphoscoliosis leads to a left shift and mitral configuration; correction is made by rotation into the left anterior oblique view. Left convex kyphoscoliosis produces a right shift and an apparent widening of the aortic shadow; correction is made by rotation into the right anterior oblique view. Kyphosis causes a foreshortening, which is revealed by a lateral view. A funnel chest causes a left shift and a decrease in density, findings which are understood when observed in a lateral view.

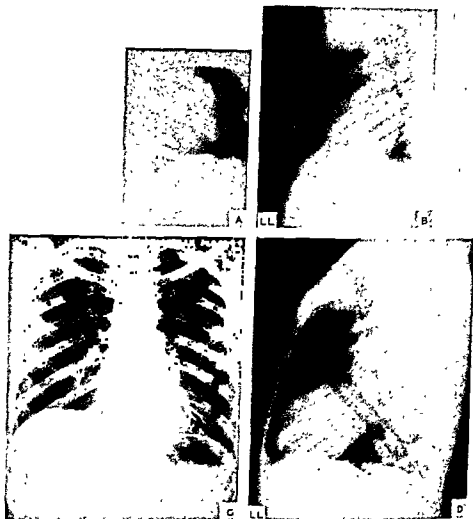
**Trauma. CONTUSION.** In only a few instances of contusion have roentgen studies been made. In one case with traumatic rupture of the aortic valves, in a young man, an aortic configuration and enlargement of the left ventricle were demonstrated.

Recent experimental studies upon animals have revealed very interesting findings. Similar findings may perhaps be expected in man if serial studies are made, starting as soon as possible following trauma to the chest. Contusion of the chests of rabbits and of cats by a stroke of the reflex hammer leads to a sudden spherical enlargement of the silhouette, with an average area increase of 23% for rabbits and 12% for cats, the degree of dilatation is proportional to the degree of trauma. There ensues a fall of arterial and a rise of venous pressure. The very marked electrocardiographic changes rule out mere reflex effect. The regression is found to occur after several minutes or even hours but may be prolonged and followed by a secondary dilatation. In animals which have been sensitized by horse serum, a peculiar silhouette results following trauma; a constriction appears around the lower pole region. The electro-

inferior portion of the septum. Furthermore, the atria show marked dilatation. Coronary occlusion, however, is not noted.

**FOREIGN BODIES.** Stab and gunshot wounds are specifically to be considered. A hemorrhage into the pericardium is of great significance and often is an indication for surgical intervention. (The appearance is dealt with in chapter XI.) Though many surgical operations on the heart have been successful, approximately one-fourth of these patients develop a postoperative pericarditis and mediastinopericarditis. A ligature of a coronary artery branch may lead to an infarct. Thus late abnormal roentgenological findings are to be expected.

Missiles may reach the heart directly or by way of the venous system and may be dislodged from the heart into both the lesser and the greater circula-



tion It therefore becomes necessary to submit the patient in whom an intracardiac or abdominal missile has been diagnosed to another roentgen study immediately preceding the scheduled operation Further, the chest must be included in the roentgen examination when, following a penetrating abdominal wound, the missile is not found in this region. At least fifty percent of all

patients with a metallic foreign body in or upon the heart suffer from pain, dizziness, oppression, dyspnea, and attacks of irregular heart action. Often the presence of these foreign bodies has not been suspected clinically and patients have been considered neurotics and malingerers. In addition, the localizing diagnosis of these missiles is important because the site of entrance is not a reliable criterion for their final resting place. Migration of these foreign bodies has been roentgenologically demonstrated not only from the inferior vena cava and hepatic veins into the right heart but also backward from the right atrium into the hypogastric and femoral vein. They may also migrate from the right heart into the bifurcation or into one of the main branches of the pulmonary artery; or from the left ventricle into the carotid, subclavian and femoral arteries. Usually, however, the missiles become lodged in the heart or in its immediate environment. Location within a cavity is indicated by a dancing, whorl-like movement, "like a pill shaken in a box." Marked movements, occurring spontaneously or induced by changes in the position of the body are necessary for the localization of a foreign body in the cavity of the heart. Peculiar movements are often observed, for instance, a wandering motion along the inner lower margin of the left ventricle; then with systole an upward movement along the interventricular septum and slowly down along the upper and outer margin toward the apical region during diastole. Further, the foreign body may remain quiescent during several cardiac cycles and then

be observed to "like a superior and inferior vena cava. Sooner or later these peculiar movements cease and become dependent upon the action of the heart and respiratory influences, indicating that the missile has become embedded. As a whole, intramural foreign bodies show a pendulum movement. Differences in type of movement are explained by the fact that the fibers in different layers of the ventricular muscle extend in different directions. Bullets may partially project into a cavity and receive an impulse from the blood current. Finally, the movement is determined by the topical site. Those missiles which are fixed close to the base of the heart move extensively with each systole to the left and caudad, the extent of the amplitude being 1 to 3 cm. This is proof of the wide physiological excursion in the region of the atrio-ventricular septum. Fixation in the left or anterior wall shows a systolic movement to the right and caudad. This is substantiation of the physiological rotatory movement of the heart. Were the heart a spherical body, there would be no difficulty in clearly distinguishing between intra- and extracardial location. The presence of grooves on the surface, however, often makes this differential diagnosis impossible in the case of small metallic foreign bodies. Free shifting along the surface of the heart is observed in the location of a missile in the pericardial cavity; especially in the presence of an effusion. Later, organization takes place and the diagnosis as to superficial, intramural, intrapericardiac or extrapericardiac site becomes difficult. Transmitted cardiac pulsations are noted in the case of missiles with a mediastinal anchorage.

ROENTGEN RAYS. The heart and the pericardium might be expected to

undergo damage when a sufficiently large amount of radiation is given over the chest.

The experimental results which were obtained on animals are interesting but a critical persual of the available data clearly reveals that the amount of irradiation used in these experiments was much greater than is ever used on man. This applies particularly to small animals, the deeper tissues of which receive a very large percentage of the surface dose.

In the course of several months, dogs and sheep were given a total of 5 to 19 erythema doses (E D) over the entire chest. Nearly all the animals died. The anatomical findings consisted of pericardial effusion, marked dilatation of the heart affecting chiefly the right side, edema of the connective tissue, varying forms of muscle fiber degeneration, capillary hyperemia, hemorrhagic infiltration of the right atrium, thickening of the coronary artery branches and degeneration of the sino-auricular node, although little change was observed in the bundle and its branches. It is not surprising that an increase of the heart rate occurred and that marked electrocardiographic variations were observed, such as inversion of T waves in one or more leads and in some instances auricular fibrillation and flutter.

The precordial area of rats and rabbits was given 2 E D. within twelve weeks. Degeneration and necrosis, varying degrees of inflammatory reaction and early interstitial proliferation were found. Bronchitis and pleuropulmonitis were present as a direct result of irradiation. Chromatolysis and lipoidosis of the ganglions and edema of the nerve trunks in the mediastinum as well as destruction of the lymphoid tissue was demonstrated. Thus it is more than probable that cell disintegration and bacterial infection are responsible, at least in part, for the microscopic findings in the heart muscle.

The available experimental data indicate that the heart muscle is less sensitive to irradiation by roentgen rays than are lungs and pleura. The application of a large dosage is followed by inflammatory and degenerative changes in tissues other than the heart muscle, but the circulation of products of disintegration and a superimposed bacterial infection may well occasion toxic changes in the heart muscle itself. A very large amount of irradiation, however, does cause direct damage to the heart muscle.

Other experiments were directed toward the influence of the roentgen rays upon blood vessels. The right extremities of dogs were irradiated and later the animals were sacrificed. The vessels in the extremities were injected with a contrast medium and comparative roentgenograms were made upon frozen sections of all extremities. Two E D. given within three months had no effect. Following the application of 3 E D. in 8 weeks, a narrowing of the vessels was noted; 4 E D. given within 6 months, a narrowing of the vessels was noted; the number of visible vessels, which had been given over a period of 6 months.

The extremities of dogs were irradiated with  $\frac{1}{4}$ -3 E D., the animals sacrificed and the reaction of arterial strips was tested in respect to Adrenalin and galvanic current. It was found on comparison to be different from the reaction of the arterial strips of the non-irradiated left extremities.

In man the heart is exposed to roentgen rays during the irradiation treatment of breast, mediastinal, pleural and lung tumors. The material which has been

studied from the standpoint of possible damage to the heart is too meager to permit of definite conclusions. One author reports the case of a patient with mediastinal lymphogranuloma; 4 series of radiation treatment were given during one year, each consisting of 10 areas. Each area received 2 Sabouraud doses through 0.8 cm. Al. The postmortem examination disclosed the tumor to be completely sclerosed. The epicardium was covered with fibrinous material. The heart



FIGS 110, 111—19 yrs, F. Pneumonia at age 11. Productive cough for past 4 years. Pulmonary abscess diagnosed at age 18. Bronchoscopy: foul purulent exudate from abscess in rt lower lobe. Ant view: rt cardiac border cannot be determined. An almost spherical shadow measuring about 8 cm. in diameter is noted in mesial basal portion of rt lung field, regularly outlined, with shifting fluid level. Rt half of cardiac silhouette resembles a pneumopericardium. Rt lat view: fluid collection centrally located. Roentgenologically, an empyema of mediastinum was first considered. Bronchoscopy and surgery made pulmonary abscess probable. Microscopic: Dermoid cyst.

muscle showed, in parts, different types of degeneration such as occur in many infectious-toxic states. In addition, a peculiar ballooning of the sarcolemmal sheaths was noted and also necrosis of the myoplasm and plasmoptysis to such a degree that the sarcolemmal sheaths appeared empty. Another investigator cites the case of an individual who had a right sided mediastinal tumor which was later ascertained to be a teratoma. He first received  $4 \times \frac{1}{10}$  E.D. and then twice  $4 \times 30\%$  E.D. over 3 areas through 0.05 cm. Zn and with a 36% depth dose. Four years later the treatment was repeated with  $2 \times 40\%$  E.D.; this was followed by irregularity of the pulse, dyspnea, pericarditis and pleuritis, right lower lobe collapse and cardiac failure. Another treatment was given.

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other authorities have reported their microscopic findings in a series of 10 cases which had been irradiated over the precordial region during the course of treatment for thoracic and intrathoracic malignancy. Ten other cases which received no direct irradiation and four with malignancy but without irradiation were chosen as controls. The type of irradiation was characterized by 200 kv., 0.5 mm. Cu,  $\lambda$  0.16; the skin received 40-140% E.D., the tumors 70-140% E.D. In two cases radium was applied in the esophagus. The hearts were thought to show definitely more pathological changes than the controls and these changes consisted of interstitial fibrosis, hyaline and fatty degeneration of muscle fibers with occasional breaks in their continuity, and edema of the heart wall. The value of these conclusions is, however, limited by the fact that 6 out of the 10 cases were beyond the age of 40 and that, in addition, data are lacking regarding the findings in the coronary arteries. Such studies should be repeated with limitation to younger age groups. On the other hand, the histological examination of a heart which had received  $2\frac{1}{2}$  E.D. during a

five weeks' course of saturation treatment for mediastinal neoplasm and which was studied 3 weeks later, revealed the absence of any pathological changes.

A critical perusal of the available material leads to the conclusion that microscopic changes which were thought to be characteristic and specific for damage by roentgen rays do not exist. The proof of such damage rests upon the appearance of cardiovascular failure and changes in the electrocardiographic tracings, especially negativity of T waves, following irradiation over the heart area.

The type and amount of irradiation in man which brings about a general systemic effect and depression is accompanied by a fall in blood pressure which may continue for a few days and which has been reported to be followed by another similar phase between the 25th and 35th day and to be followed occasionally by even a third decline between the 50th and 70th day. Such periodic alternations in the biological effect of roentgen

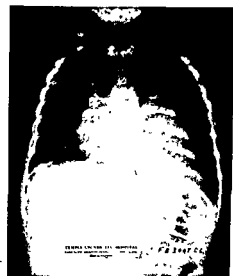


FIG 112—21 mos, M. Aspiration of peanut fragment 3 weeks previous, followed by cough and vomiting. Thoracocentesis 3 days previous elsewhere. Cardiac dullness to rt. Tachycardia. Ant. view: partial collapse lt. lung, lt.-sided pneumothorax, shift of mediastinum to rt. Course: improvement following removal foreign body. Courtesy Dr C. L. Jackson, Temple University Hospital, Philadelphia.

rays are well known, for instance, in the course of the skin erythema. The fall in blood pressure is greatest in cachectic patients and in those irradiated over the abdomen. A fall after a treatment with longer exposure is perhaps accounted for by the relaxed position of the patient during this prolonged irradiation.

A case has been reported in which irradiation of the bones for polycythemia was followed first by auricular flutter and 10 days later by coronary thrombosis. This was anatomically verified and was causally related to the irradiation. It should be remembered, however, that polycythemia in itself favors the development of vascular thrombosis. Irradiation of the neck and upper mediastinum for thyrotoxicosis has in a few instances led to precordial and retrosternal pain, fever, and E.C.G. changes of the type associated with pericarditis; recovery occurred within a few days.

Cardiovascular disturbances have often been noted with the Coutard type of protracted treatment for tumors of the pharynx and larynx. The large total amount of irradiation as well as a possible effect on the carotid sinus must be considered. It was observed that several individuals developed aneurysmal dilatation in the region of the carotids two years after this treatment. The effect is said to be proportional to the total amount and independent of the daily dose. The disturbances take place in the second and third week of treatment. The systolic and pulse pressures are lowered, tachycardia, embryonic and irregular rhythms are noted and the heart sounds become of low intensity. The E.C.G. in a series of 47 cases showed lowering of the voltage of the T deflections 8 times, and a depression of the S-T segments with a lowered T in 23 instances. Cardiopaths develop bronchopneumonia as a frequent complication, especially during the time of the radioepithelitis.

It is difficult to evaluate the direct and indirect effect of the rays in these cases.

**Tracheobronchial Stenosis.** The effect of prolonged tracheal stenosis on the heart size in man is not clearly understood. It is doubtful that stenosis caused by goiter necessarily leads to cardiac hypertrophy and dilatation. It is possible that emphysema plays a rôle. Concomitant toxic influences must likewise be considered. Observations referring to larger series are of an earlier date and while toxic conditions were excluded on clinical grounds yet basal metabolism determinations were not available. No definite correlation was found between the cardiac affection and the degree and duration of the stenosis, and post-operatively there was no demonstrable change in the heart size.



FIG 113—Middle aged, M Chronic tuberculosis for several years *Ant view*—extensive productive infiltration in both cranial-lung fields with marked shrinkage. Hilar structures displaced cranial. Vascular shadows toward caudal lung portions as well as rt. mediastinal border straightened. From Roesler, H, in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System, ed. by Ross Golden, 1936, p. 229. Courtesy Th. Nelson & Sons, New York.

Tracheal stenosis with a valve mechanism was produced in animals in such a manner that inspiration was embarrassed while free expiration was permitted, a moderate enlargement of the cardiac silhouette resulted and, when the animals were sacrificed, the right heart chambers were found to be dilated.

A marked degree of tracheal stenosis in connection with the inspiratory pull of the diaphragm and the elevation of the chest leads to a retraction of the

TABLE—PNEUMOTHORAX, RESPIRATION AND MEDIASTINUM

Type of Pneumothorax	Phase of Respiration	Direction Toward Which Mediastinal Shadow Moves	Alterations in the Collapsed Lung	Remarks
<b>I Closed</b>				
A) With Negative Pressure	Inspirium	Pneumothorax	Larger	
	Expirium	Normal Lung	Smaller	
B) With Positive Pressure	Inspirium	Pneumothorax, or Immobile	0	
	Expirium	Normal Lung, or Immobile		Depression of the Diaphragm
<b>II Open</b>				
A) With Bronchial Fistula				
1) With Wide Open Communication	Inspirium	Normal Lung	0	} Slight Degree of Displacement
	Expirium	Mid-line		
2) With Valve Effect, Permitting Inspiratory Entrance	Inspirium	Immobile		
	Expirium	Normal Lung	Smaller	
B) With Wide Chest-wall Fistula	Inspirium	Normal Lung	Smaller	
	Expirium	Pneumothorax	Larger	Extreme Degree of Displacement

lower chest. This, in a lateral view, gives the appearance of a crossed profile and the underlying mechanism must be differentiated from that which is responsible for a crossed profile in the presence of chronic mediastino-pericardial disease.

Constrictions in the trachea are likely to produce a valve-like mechanism. When the heart is compressed and, thereby, acutely tamponaded, a squeezing out of the heart, occurs. The cardiac silhouette attains normal size during the phase of inspiration. Such an obstructive

effect was experimentally produced in dogs and a diminution of 7-10% of the silhouette area was observed. Bronchial stenosis of one side, or predominating on one side, leads to a respiratory shift of the mediastinum. The rapidity and degree of this shift depends upon the degree and location of the stenosis, upon the mobility of the mediastinum and upon the depth of the inspiration. Complete stenosis of a main bronchus shows this shift into the affected side with quiet inspiration and an accentuation of the shift with deep inspiration. Partial stenosis of the bronchus of the posterior (lower) lobe shows no, or only a minimal, shift of the cardiac silhouette; but there is a marked shift in the presence of a complete stenosis. When an acute obstructive unilateral emphysema results, as observed for instance in the presence of foreign bodies, the mediastinal shadow appears displaced away from the affected side. Secondary atelectasis of larger lung areas causes a deviation of the mediastinal shadow toward the affected side.

**Pleuropulmonary Disease. INTRODUCTORY REMARKS.** Various pathologic chest conditions may cast a strain on the right heart. Faulty respiration, in the wide sense of the word, is frequently the etiologic factor. Reference is made to diffuse narrowing of the bronchial tree (asthma, chronic bronchitis), tracheal stenosis, kyphoscoliosis, extensive pleural adhesions, emphysema, extensive parenchymatous destruction, pneumoconiosis, obliterative vascular process, pulmonary embolism, and paralysis of the muscles of respiration. The roentgenologic study is significant for two reasons. First, it is clinically impossible to determine heart size in the presence of several of the conditions just referred to. Second, one may make an important observation during fluoroscopy, i.e., an inspiratory increase and an expiratory decrease of the silhouette. Since this observation seems to be a partial clue to the problem of right heart strain and failure, and since the functional disturbance involved seems to be unknown to pathologists and inadequately appreciated by clinicians, a short discussion is appropriate. The lungs are made up of elastic and non-elastic elements. They may be likened to the structure of a garter whose weave shows an interlacing of non-elastic hemp and elastic rubber. The latter furnishes the distensibility, provided the non-elastic elements are plaited at the rubber's state of rest. When the rubber disintegrates, the unplaited garter occupies a greater length and will not yield to traction. The mediastinum is held in position by the normal lungs as by two elastic cables, acting as physiologic-brake factors. This brake factor is abolished as soon as air is interposed between the heart and the lungs, such as is the case with a pneumopericardium or with a pneumothorax, and consequently the type of cardiac pulsations changes immediately to a more vigorous action. On the other hand, an augmentation of this brake factor may be observed. When one lung is extensively sclerosed, it loses its elasticity and acts as a rigid cable. The energy that would normally be used to distend a normal lung is exerted from the chest wall as direct traction and there results a respiratory shift of the mediastinal shadow. When both lungs lose their elasticity as a result of sclerosis, infiltration or loss of the elastic system, something must yield with the inspiratory distension of the chest cavity, and consequently there results an inspiratory *distention* of the cardiac silhouette. This distention is noted in the various views. Simultaneous with this distention, the amplitude of pulsations tends to diminish. The

silhouette decreases in size during expiration. These changes are always best noted along the border of the right atrium. The blood pressure level varies. It is lower during inspiration, higher during expiration, and there is a paradoxical pulse in the periphery. Whenever inspiration coincides with cardiac systole, the latter is inhibited. It is conceivable that this interference, continuing over a period of years, contributes to the development of right heart hypertrophy and, finally, to failure. Further studies are required in order to establish the sig-



FIG 114—33 yrs, F Pneumonia, tuberculosis, pleurisy in past. Dyspnea and palpitation Asthenic type Moderate cyanosis. Fibrous type of tuberculosis and bronchiectasis Hard cervical and axillary nodes palpable Heart not enlarged P<sub>2</sub> accentuated ECG. n.s.r, right axis dev *Ant view*: cardiac silhouettes of vertical type; prominence of pulmonary arch Extensive calcification of mediastinal lymph nodes Pulmonary fibrosis and bronchiectasis, predominantly at lt Group of large calcified lymph nodes of extra-thoracic localization

nificance of intrabronchial, intrapleural and mediastinal pressure changes that may be associated with marked silhouette changes during the respiratory cycle. Another point deserves attention. It has been demonstrated by means of animal experimentation that the pressure in the pulmonary artery and right ventricle are closely related to the degree of pulmonary distention. Apparently the stretching of the capillaries beyond a certain point increases the resistance to blood flow. Hence pulmonary distention produces hypertension in the pulmonary artery circulation, and greater work is required from the right ventricle

**PNEUMOTHORAX.** The important findings are the respiratory changes of the mediastinum and the appearance of changed ac-

tivity along the cardiac silhouette. Different types of pneumothorax cause various respiratory movements of the mediastinum (This is outlined in the table). Marked differences in pressure may lead to a herniation of one lung into the opposite side. This is made possible by the presence of the weak areas in the mediastinum, one ventrad and cranial, the other dorsad and more caudad. The shadow of the mediastinal pleura appears as an arclike dense line in one or the other lung field. It must be differentiated from the arclike appearance of the distended pericardium in the presence of air in the pericardial cavity (this is discussed in chapter XI). Lateral views commonly reveal a dorsal displacement of the cardiovascular silhouette in the presence of a pneumothorax. The volume of the heart has been determined prior to and following pneumothorax, and a significant diminution in size has been frequently observed. Anatomical

and dilatation in a few cases, he pulmonary artery may diminished and therefore

only rarely has a pulsatory, systolic expansion been observed. More often one observes a vibratory movement, the speed of which is greater than the heart rate.

With the chest wall closed, the heart is embedded between the lungs, which

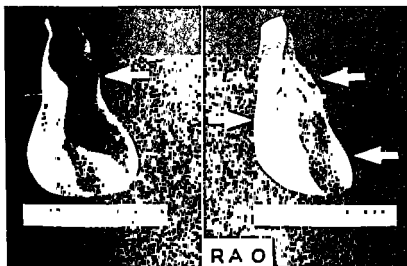


FIG 115—40 yrs, M Emphysema. Roentgenologic, volumetric reconstruction. *Ant view*: globular heart without enlargement, prominence of pulmonary conus and artery (arrow). *Rt ant obl view*: normal findings along dorsal border, no enlargement of lt atrium (lt arrow). Cranial portion rt ventricle prominent (conus arteriosus), (lt upper arrow). Rt ventricle, caudal portion (lt. lower arrow). (Courtesy Prof Dr G G Palmieri, Bologna, Italy)

exercise traction upon its entire surface. By opening the chest wall or by introducing air or gas into the pleural cavity this effect is eliminated, the restrictions on the heart movements cease and large pulsations result. It is necessary, however, that the air shall reach the mediastinal surface, accumulation along the parietal surface apparently does not cause such changes. When the pulsations of a dog's heart are compared, first under fluoroscopy and then in the open chest with collapsed lungs, an enormous difference is noted; when in the recumbent position, the heart is seen to fall dorsad and to the left during diastole and to spring ventrad and to the right with each systole and a twisting rotation is observed. As soon as the lungs are fully inflated and the heart is supported on both sides, the rotation of the organ ceases. A further proof of this assumption is afforded by the following observation in man. During the inspiratory lateral displacement of the heart the partially collapsed lung may move more slowly than does the cardiac shadow, contact therefore results and at once the abnormally large pulsations cease. During expiration, the cardiac shadow and lung separate again and at once the normal pulsations are replaced by the abnormal type. These abnormal pulsations are observed only on the side of the pneumothorax and are much more marked when the pneumothorax is left sided. As compared with normal activity these pulsations are larger in

amplitude, they seem to take place at a greater speed, even in the absence of tachycardia and are peculiarly flopping in type. Atrial and ventricular movements can be well differentiated. In addition, there is a marked vertical, heaving component in all the visible movements. Rotation is undoubtedly marked; this perhaps accounts for the rather surprising fact that a kymographic study registered the outward movement mainly as systolic and the inward movement mainly as diastolic.



FIGS 116, 117—45 yrs, M Asthma and emphysema since age 40. Marked weight loss. Productive, purulent cough. Clubbing, cyanosis, edema, hepatic enlargement and ascites. Moist sibilant rales over lungs. Blood in sputum.  $P_2+$ , B.P. 115/60 E.C.G. n.s.r, right axis dev,  $T_2$  diphasic. Emphysema, bronchiectasis, cor pulmonale. Ant view cardiac area globular, prominence of pulmonic arch. Heavy density throughout both lung fields, extending along broncho-vascular trunks. Rt ant, obl view, marked bulge ventrad, corresponding to cranial and ventral portion of rt ventricle (conus arteriosus) (wh arrow). Prominence of pulmonic knob into posterior mediastinum (bl arrow). Lt. atrium not enlarged. Orthodiagraphic measurements L 14.7 cm, B 12.4 cm, T 13.6 cm,  $T_D$  11 cm,  $\frac{T_D}{T}$  80.8% (norm av 73.0%),  $\frac{T_D}{L}$  74.8% (norm av 66.3%). Course: severe cardiac

failure, death within three months. Right ventricular hypertrophy and slight enlargement increase the depth of the heart as the measurement reveals. They also cause prominence of mass of heart cranial and ventrad which is best visualized in oblique and lateral views.

A marked displacement of the mediastinum, as may occur in the presence of a pneumothorax with positive pressure, leads to signs and symptoms of cardiac failure and the roentgenological findings may prevent the clinician from making an erroneous clinical diagnosis of pulmonary disability.

Marked differences in the volume of both lungs lead to a displacement of the mediastinal shadow. Among rare conditions of an increase of one lung volume, congenital in origin, and usually observed only in children, are true hyperplasia and diffuse cystic or bronchiectatic degeneration. A pathological decrease in the volume of one lung is usually acquired, though congenital

malformation exists. In such conditions as are commonly typified by tuberculous sclerosis, the assumption that mediastinal displacement is necessarily caused by a pleural affection, is certainly erroneous. Marked displacement in the presence of pulmonary pathology does not necessarily indicate an adhesive pleural process and is therefore no contraindication to a therapeutic pneumothorax. The ordinary roentgenologic technic, when employed on patients with advanced fibrothorax, will frequently fail to disclose the location and structural state of the heart, the great vessels and the pulmonary circulation. Successful visualization is obtained in such instances by the rapid intravenous injection of a concentrated solution of diodrast and by taking the roentgenogram at the moment of opacification of the structures to be visualized. These displacements are especially marked in younger people where the healthy lung compensates by expansion and hypertrophy. The presence of massive atelectasis in one lung leads to a displacement of the mediastinal structure toward the diseased side. Pneumonia of one whole lung or only of its posterior (lower) lobe causes a similar effect in early childhood but the finding of displacement is not constant in adults.

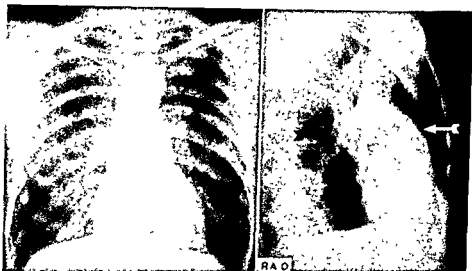
**TUBERCULOSIS.** Patients with certain forms of tuberculosis of the lung may present signs and symptoms similar to or identical with those found in primary cardiovascular disease. In the exudative type of tuberculosis and generalized, hematogenous dissemination of the disease a toxic condition is found with pallor, cyanosis, tachycardia, instability of the pulse and low blood pressure. In the chronic fibrotic type that is characterized by pleural thickening, sclerosis and emphysema, and by displacement, kinking and denudation of the large vessels, there occurs a predominantly mechanical embarrassment. This leads to palpitation, oppression and air hunger on exertion, murmurs and often a marked accentuation of the second pulmonic sound may be heard.

Respiratory displacement has already been mentioned. A fibrotic retraction in the anterior (upper) lobes, often combined with compensatory basal emphysema, results in a straightening of the borders of the silhouette, in addition, the main pulmonary vessels are displaced cranial and become kinked, and the vessel shadows of the caudal lung areas run straight like extended fingers. A marked degree of left anterior (upper) lobe fibrosis may lead to an undue prominence of the pulmonic arch of the silhouette. A complete displacement of the heart to the left and dorsad leads to rotation and kinking of the aortic arch. Serial studies were published reporting observation of some enlargement of the cardiac silhouette to the right as fibrosis and emphysema developed, and occasionally also in the course of acute infiltrations.

It is a common impression that the cardiac silhouette is small in tuberculosis of the lungs. Such a general statement is not acceptable; it is not reasonable to assume that tuberculosis with its different clinicoanatomical forms exerts a uniform influence on the heart. The argument that a certain constitutional type should predispose to infection with tuberculosis is likewise hardly acceptable since constitution, in the anatomical sense, does not play a dominant rôle in the development of tuberculosis. In some instances postmortem experience shows small hearts having a total weight below normal, particularly in patients with miliary tuberculosis or caseous pneumonia. This is to be expected with a



amplitude, they seem to take place at a greater speed, even in the absence of tachycardia and are peculiarly *flopping* in type. Atrial and ventricular movements can be well differentiated. In addition, there is a marked vertical, heaving component in all the visible movements. Rotation is undoubtedly marked; this perhaps accounts for the rather surprising fact that a kymographic study registered the outward movement mainly as systolic and the inward movement mainly as diastolic.



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toxic or wasting disease. In other instances although the total weight is still within normal limits, a separate dissection of the right and left ventricles reveals a functional predominance of the former. This may be observed with the chronic-cirrhotic type. Also there has been noted a moderate degree of right ventricular enlargement, but never is there present an excessive degree of hypertrophy. There seem to be three explanations for the erroneous general statement of a small heart. First, chest films are usually taken on deep inspiration; second, the comparison of the actual roentgenological heart size is not made against adequate standards, i.e., with the same physical characteristics, such as muscular development, chest circumference and the like; third, the diagnosis of a small heart may be made only when the depth development and the examination in the recumbent position confirm the results obtained by the examination in the anterior view and upright position. With these criteria in mind, it may be stated that about two-thirds of all these hearts are of adequate size and perhaps less than 10% are actually small.

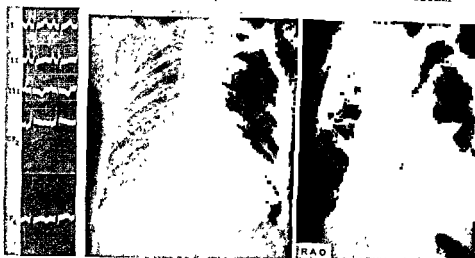
**ALLERGIC BRONCHIAL ASTHMA; EMPHYSEMA.** The cardiac silhouette remains singularly free from enlargement in asthma. As a matter of fact it seems to tend toward lower limits, even when we consider cases without low position of the diaphragm. But, it should be remembered that asthmatics also have a tendency to be underweight, often exhibit a flabby, poorly developed muscular system, and always have a poor respiratory action even in periods of remission. During severe attacks one finds prominence of the pulmonic arch and the lung vessels, and inspiratory distention of the silhouette. The heart volume in one such case was determined to be 650 cm<sup>3</sup> during inspiration and 530 cm<sup>3</sup> during expiration. That the faulty respiratory mechanism seems to have an effect on the heart, is shown by an anatomic study on 70 cases where the chief clinical diagnosis had been bronchial asthma. Only two cases were complicated by hypertension. The quotient of actual heart weight to body weight was compared with the quotient of expected heart weight to body weight. 15 cases revealed normal figures, 9 were below normal, and 46 were above normal. To what degree associated emphysema might have influenced the development of cardiac hypertrophy is unknown.

In true emphysema, the lungs are deprived of their elastic elements and the force which causes physiological expiration is diminished or disappears. The velocity of the blood through the lungs is not altered; some dilatation of the pulmonary artery and its branches, often combined with atheroma and with some right sided hypertrophy, makes the presence of hypertension in the lesser circulation probable. The poor action of the diaphragm removes a powerful

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spaces. Normal acini were not found. The small bronchi and bronchioles were dilated, showing cell infiltrations and plugging by aggregates of sloughed cells. The vessels showed a hyperplastic type of sclerosis with diminution of their lumina.

*Comment:* An instance of chronic cor pulmonale. The chief pulmonary lesion was a congenital polycystic disease. Occupational harmful influences probably played an additional rôle. Neither chronic pulmonary suppuration nor syphilis seemed to be etiological factors, and vascular sclerosis was not the predominant pathology. The clinical picture was governed by inadequate oxygenation of the blood. There was present strain on the rt ventricle, hypertrophy and dilatation affecting particularly its outflow tract.



**Figs 118, 119**—60 yrs, M History Working as a brass finisher, the patient had been inhaling brass dust for a period of 47 yrs. He had been coughing for the past 15, dyspnoic for the past 5 yrs, and orthopnea had developed recently. Precordial pain accompanied paroxysms of cough. Findings: There was no fever, the skin was dusky, and clubbing was noted. There was no evidence for congestive failure in the greater circulation. Inspiratory and expiratory wheezing and moist-crackling rales were heard over the lungs. The heart was moderately enlarged, the rate was 125/min, a gallop rhythm was heard, and  $P_2$  was accentuated. A paradoxical pulse was noted. BP 140/95. The blood showed hb. 16 gms, rbc 6.6 mill, Wassermann test positive. The fundi showed a minimal degree of arteriolosclerosis. Circulation time for arm to tongue (saccharin) was 18.5 sec., and arm to lung (ether) 11.3 sec. ECG (next day) Nsr, rt axis deviation,  $T_1$  negative.

**Ant view (11-26-40)** The lung fields show a diffuse, finely mottled and partly stringy type of density. These changes obscure, to a certain degree, the cardiac borders. The heart is moderately enlarged, and some prominence of the pulmonary arch is noted (1). On fluoroscopy the diaphragm was noted to move very poorly. **Rt. ant obl view:** Small bronchiectatic cavities are noted in the rt. lower lung field. The heart reveals no prominence corresponding to the lt. atrial area (2) but, ventrally, the rt. ventricle bulges (3).

**Course** Oxygen inhalations and codeine sulphate gave relief and prolonged bed rest was beneficial. Digitalis seemed to have no appreciable effect. The pulmonary findings remained essentially unchanged, the gallop rhythm disappeared and the BP remained at about 110/80. The vital capacity was 23%, the circulation time for arm to tongue (saccharin) was 16.5 sec and for arm to lung (ether) 7.0 sec.

**Postmortem (2-18-41, on embalmed body):** The heart weighed 580 gms and was considerably enlarged. The rt. ventricle was larger than the lt., its lumen was greatly dilated, its wall measured 1.3 cm in thickness, and, corresponding to the outflow tract, the wall was so prominent as to be almost aneurysmal in extent. The lt. ventricle was dilated, its wall measured 1.6 cm. The orifices were normal, the diameter of the pulmonary orifice was 3.0 cm and that of the aortic orifice, 2.6 cm. The coronary arteries were

lobes so that the surface showed a pebbled appearance. The cysts measured from 0.1 to 0.3 cm in diameter. About the lateral and ventral borders bullous distentions were present, the largest of which measured 2.5 cm. in diameter. The bronchi and arterial vessels showed nothing of significance. There was present acute and chronic congestion. Microscopic. Besides the supportive tissue and the blood vessels, the lung tissue consisted of very irregular spaces of all sizes which were lined with tall columnar epithelium resembling bronchial mucosa. Dense fibrous connective tissue surrounded the epithelial

toxic or wasting disease. In other instances although the total weight is still within normal limits, a separate dissection of the right and left ventricles reveals a functional predominance of the former. This may be observed with the chronic-cirrhotic type. Also there has been noted a moderate degree of right ventricular enlargement, but never is there present an excessive degree of hypertrophy. There seem to be three explanations for the erroneous general statement of a small heart. First, chest films are usually taken on deep inspiration; second, the comparison of the actual roentgenological heart size is not made against adequate standards, i.e., with the same physical characteristics, such as muscular development, chest circumference and the like; third, the diagnosis of a small heart may be made only when the depth development and the examination in the recumbent position confirm the results obtained by the examination in the anterior view and upright position. With these criteria in mind, it may be stated that about two-thirds of all these hearts are of adequate size and perhaps less than 10% are actually small.

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factor from the circulation; artificial elevation of the diaphragm by elastic bandage or pneumoperitoneum actually improves the circulation. Although emphysema unfavorably affects the respiration, the cardiac factor is seldom pronounced until late in the disease or unless there is present heart disease of other etiology. Hypertension is actually found to be a common complication. When heart failure occurs on the basis of emphysema, it is right heart failure with normal rhythm, and it is terminal. The postmortem findings are frequently those of right ventricular hypertrophy and dilatation, but this is apparently not an absolute rule, and the reason for the heart muscle remaining thin throughout in some instances is unknown.

When one limits the roentgenologic study to the anterior view, failure to disclose prominence of the pulmonic arch and conus of the right ventricle will frequently result. It is imperative, therefore, to rotate the patient into the right anterior oblique view. Roentgenologically, perhaps one-third of the patients do not have any cardiac enlargement, nor do their lung vessels reveal changes. There is a tendency, however, to underestimate the heart size unless the oblique and lateral views are included in the study, and the use of the cardio-thoracic ratio is certainly misleading. Enlargement to the left is due to either right or left ventricular preponderance, in the latter case it is usually in connection with hypertensive and atherosclerotic disease. Great enlargement of the right heart is rare. A subdivision at the right lower silhouette border just above the right cardio-hepatic angle indicates that the right ventricle participates in the formation of the right border. When enlargement is caused by the right ventricle, one observes the following in the oblique views: Left: the projection and curvature of the ventral border exceeds that of the dorsal. Right: the cranial-ventral contour frequently bulges, the bulge representing the pulmonary artery in the upper portion and the conus of the right ventricle in the lower. A line of junction between these two elements cannot be identified. A ventral bulge further caudad indicates involvement of the body of the right ventricle. The depth development of these hearts is considerable. While the average ratio of  $T_D/T$  equals 73%, it was found to be 80% for a group of hearts in emphysema. The left atrium plays no rôle in this increased depth because this cavity remains unaffected in non-complicated emphysema, and it is this which accounts for the freedom from auricular fibrillation. The middle left arch, as visualized in the anterior view, is often though not always prominent. Sometimes its prominence is obscured by the shadow of the enlarged left main branch of the pulmonary artery. In what relative degrees this projection is composed of the pulmonary artery proper and the conus of the right ventricle, is a question which has been approached by correlated roentgenologic-anatomic studies and by visualizing the heart and the thoracic blood vessels by the injection of a concentrated solution of diodrast. Variable findings were obtained by the former studies: the prominent arc could be accounted for either by the pulmonary artery alone, or predominantly by the conus, or by the pulmonary artery in the upper two-thirds and by the conus in the lower third. A contrast study has been carried out for one case which revealed that the conus did not participate but that the whole arc was formed by the enlarged pulmonary artery, which, in addition, was displaced cranial and to the left by the elongated right ventricle. In at least half

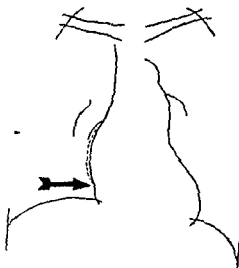


FIG 120—46 yrs. M. Pneumonia and pleurisy at age 32. Dyspnea and palpitation on exertion since then. Edema first occurred at age 39. Cough and bronchitis. Cyanosis, expiratory dyspnea, emphysema. Heart action not palpable. Lower sternal portion dull on percussion. Sounds faint,  $P_2$  acc. BP 100/65. E.C.G. n.s.r., rt axis dev. Ant view diaphragm in low position, of diminished excursion. Cardiac shadow elongated caudad. L 152, B 96, T 124, Th 252 cm. Rt. lower cardiac contour reveals subdivision (bl arrow) into longer cranial and shorter caudal portion; the former bulges laterally 0.4 cm. during each inspiration. Prominence of second lt arch (pulmonary

trudes in front. Postmortem. pneumonia, lung abscess, bronchiectasis of rt. ant lobe, purulent bronchitis, bilateral pleural adhesions. Marked hypertrophy and dilatation of rt. ventricle which forms apical portion. Width of rt. ventricle 9.5 cm., of lt. ventricle 5 cm. Atria neither enlarged nor hypertrophied. No valvular disease. Atherosclerotic changes in pulmonary branches, microscopic examination not undertaken. Incidence of rt. ventricular hypertrophy and dilatation secondary to pulmonary disease. The rt. ventricle forms the caudal portion of the rt. cardiac border. The inspiratory distention affects the thin-walled rt. atrium only. The increase in heart size was expressed by L only and the rt. ventricle caused an enlargement to the lt. and caudad.

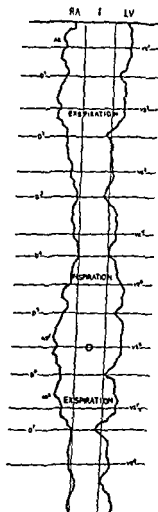


FIG 121—Emphysematous dyspneic patient. Kymographic tracing of lt and rt cardiac borders. Film moved from above downward. The diaphragm was in extreme low position, flat, almost immobile and could not cause any shift of the cardiac borders. An inspiratory widening of the cardiac shadow is noted (simultaneous lateral movement of the cardiac borders). This is followed by an expiratory narrowing of the cardiac silhouette (simultaneous, steep, medial movement of either cardiac border), vs — beginning of ventricular systole, D — beginning of ventricular diastole. From *Fortschr. Geb. Röntgenstr.*, 1934, 49, 240. Courtesy Doz. Dr. E. Zdansky, Vienna and G. Thieme, publisher, Leipzig.

of all cases the lung fields show the hilar (arterial) vessels to be prominent, and occasionally there is noted a minor degree of intrinsic pulsations. In the left anterior oblique view the left main branch of the pulmonary artery is noted to be unduly prominent, i.e., larger and denser than in normals. Finally, the inspiratory distention of the silhouette followed by an expiratory diminution, previously referred to, should be mentioned.

**PNEUMOCONIOSIS.** The substance that produces changes of significance is free silica ( $\text{SiO}_2$ ). Its particles are phagocytized and carried into the lymph channels, which become obstructed. Fibrosis is stimulated and the tissue thus formed around the bronchi and small branches of the pulmonary arteries gradually replaces the lung tissue. Extensive parenchymatous, interstitial and vascular disease explains the anatomical finding of right ventricular hypertrophy. Its muscle weight amounts, on an average, to 40-48% of the total ventricular musculature as compared with a normal figure of 36%; and the increased resistance in the lesser circulation accounts for the fact that at the age of 45-50 years the pulmonic root is larger than the aortic root. The association with tuberculous-exudative lung disease apparently

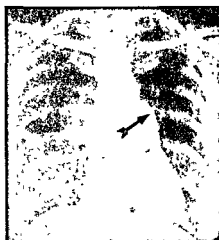


FIG 126—51 yrs, M Miner nearly entire adult life Cough, expectoration Pneumoconiosis Ant view lung fields reveal pneumoconiosis Low position of diaphragm, cardiac silhouette not enlarged. Marked prominence of pulmonary conus and artery (bl arrow) As a result of the resistance in the lesser circulation, the outflow tract of the rt ventricle, and the pulmonary artery are affected Courtesy Dr Charles F Nichols, Philadelphia

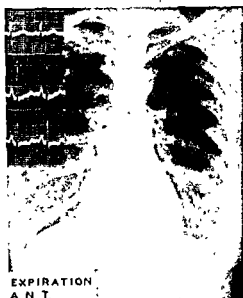
may counteract the development of right ventricular hypertrophy Out of 35 cases, who died in rightsided failure and who showed right ventricular enlargement, there were 13 who had pure silicosis, and of these 11 revealed definite right ventricular hypertrophy Twenty-two of the cases showed a combination of silicosis with tuberculosis, and of these only 9 showed hypertrophy.

Patients with severe emphysema and pneumoconiosis clinically reveal dyspnea and cyanosis and are sometimes thought to have primary cardiovascular disease. Though heart failure may be present, the two findings can often

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*Comment* In patients with emphysema—particularly when it is complicated by bronchial asthma—an inspiratory increase and expiratory decrease in heart size is noted. Whenever inspiration coincides with cardiac systole, the latter is inhibited This seems to cause the paradoxical pulse sometimes observed clinically It is conceivable that this interference, continuing over a period of years, contributes to the development of right heart hypertrophy and, finally, to failure That an actual change in the heart size takes place, is proven by observation in more than one plane The practical absence of diaphragmatic movement eliminates a major variable in this conclusion





FIGS. 122, 123, 124. Studied 7 and 5- severe, persistent. Findings: Wt 65 were noted. The cardia (pt had

the past 8 yrs. When  
were normal. Recently  
sured ether-oil enema-  
ly, and sibilant rhonchi  
rate degree of tachy-  
paradoxical pulse were present. BP 115/85.  
unted, R<sub>1</sub> relatively low, no axis dev., S-T<sub>1-2</sub>.

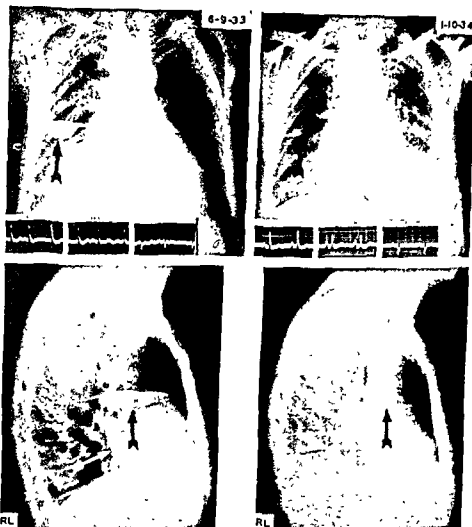
not enlarged. During auscultation, a murmur in heart size during expiration and an increase during inspiration were noted. *Lt. ant. obl. view:* Again the heart size diminishes during expiration, and increases during inspiration.

lung failure this fact should be remembered. Obliterative pleurisy permits the persistence of fissures and lacunae in the interlobar septum. Fluid can collect here and produce a well-demarcated round or oval homogeneous shadow which has been erroneously interpreted as benign or malignant tumor, gumma or exudative tubercular infiltration. Its disappearance with cardiac compensation, and its reappearance with failure proves it a small, encapsulated interlobar hydrothorax. For further discussion see chapter VI.

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## CHAPTER VII CARDIOVASCULAR DISEASE FROM THE VIEWPOINT OF ETIOLOGY

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FIGS 127, 128, 129, 130—70 yrs. M. Recently bronchopneumonia. Cough, dyspnea and palpitation. Emphysema, arteriosclerosis, bronchiectasis. Short systolic mitral murmur. B.P. 125/70. E.C.G. auricular fibrillation, lt. axis dev. R-T<sub>1</sub> slightly depressed, T<sub>1</sub> low voltage. *Ant. view*: marked cardiac enlargement without typical configuration. Sharply circumscribed oval density in rt. lung field (bl. arrow), corresponding to encapsulated fluid collection in fissure between right, upper and middle lobes. Rt. lower lung field shows moderately dense opacity, corresponding to fluid collection in fissure between right, middle and lower lobes. *Rt. lat. view*: lt. atrium not enlarged. Spindle-like fluid collections, horizontal (bl. arrow) and oblique (wh. arrow). Follow-up study 6 months later: absence of failure, E.C.G. n.s.r., otherwise unchanged. *Ant. and rt. lat. views*: size of silhouette definitely diminished. Remnant of interlobar process visible (bl. arrow).

be easily explained on the basis of a diminished vital capacity and lack of pulmonary oxygenation (anoxemia).

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## CHAPTER VIII

### CARDIOVASCULAR DISEASE FROM THE VIEWPOINT OF STRUCTURAL CHANGES

**Myocardial Disease. INFECTIOUS AND TOXIC CONDITIONS.** Such conditions are more frequent than is commonly realized. In addition to the better known etiological factors such as diphtheria, rheumatic, scarlet and typhoid fevers, pneumonia, grippe, tonsillitis, and the entity known as interstitial myocarditis, there still remain obscure factors that need elucidation. The roentgenologic study refers chiefly to an evaluation of heart size. It has been mentioned in previous chapters that findings obtained when a patient is studied in the upright position may differ from those obtained in the recumbent position. This fact applies particularly to the cases under consideration here. Patients in an infectious or toxic state are prone to suffer from peripheral vasomotor failure, and their tendency to go into a collapse is a well known clinical fact. This vasomotor deficiency is responsible for an inadequate filling of the heart when the patient is standing. Evaluation of heart size, therefore, should be carried out under optimal filling conditions, and these are present in the recumbent position. Since enlargement of the heart may not be detected in the standing position, the term "latent dilatation" has been coined. However, some degree of enlargement resulting from chronic or recurrent infectious and toxic states may be revealed even by examination in the upright position. True hypertrophy may then be noted anatomically in addition to dilatation. Physical exertion superimposed on a diseased heart muscle probably fosters such development. Advanced enlargement usually shows association with heart failure and ultimately ends in death. When the dynamics of the heart muscle have already been altered prior to the onset of diffuse muscular damage, one notes that the process of dilatation prevails for those chambers that were already under strain.

**Diphtheria.** Cardiac complications are estimated to appear in 10-30% of all cases. The type of the epidemic and the early and adequate treatment are influencing factors. Cardiac dilatation may develop rapidly, but not to an excessive degree. It is known from serial roentgenological studies that if dilatation occurs, it may commonly be expected to begin during the second week, to reach the maximum in the third week, and to regress in one to two weeks. In malignant cases, widening of the silhouette may set in as early as the third or fourth day and reach a maximum on the eighth day. The conus dilatation is quite outstanding. The linear increase may amount to not more than 1 cm. on the average with perhaps a maximum of 3 cm. Shortly before death additional increase may be observed. The dilatation in the non-lethal cases rarely exceeds 1.5 cm. for L.

The roentgenologic observation of regression of the heart size is an excellent prognostic criterion. In exceptional cases some degree of dilatation may persist for several months. Physical exertion during convalescence may bring about cardiac failure, and then an acute dilatation is observed roentgenologi-

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cally. The widening of the silhouette always seems to be associated with a diminution of the amplitude of pulsations. Widening of the vascular shadow to the right is also described.

*Scarlet Fever.* A widening of the silhouette has been observed to take place

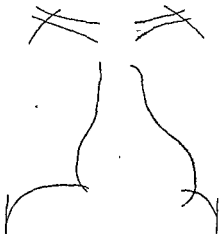


FIG. 133—26 yrs, M Health excellent up to 10 months previous, active in all types of athletics Then developed sore throat and tonsillar abscess This followed by dyspnea, weakness, precordial oppression and occasionally rise in temperature Never recovered No cyanosis. Dyspnea on effort. Heart

post-infectious myocarditis *Ant view*—moderate enlargement of silhouette L 14.2, B 10.4, T 13.8, Th 24.6 cm Amplitude of pulsations very small *rt ant obl view* (illustration omitted) Lt atrium not appreciably enlarged

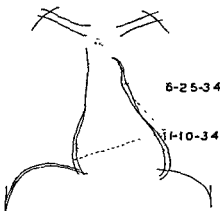


FIG. 134—16 yrs, M A few months previous streptococcal bloodstream infection during which a loud systolic murmur was heard. Referred for evaluation of cardiovascular system Normal clinical and ECG findings *Ant view* silhouette normal in size, shape and pulsations; L 11.9 cm, B 9.6 cm Patient's fist fits exactly into silhouette Course: quiet life for three months then college athletics resumed This followed by exhaustion and dyspnea Follow up study: apical thrust slightly increased, otherwise normal clinical and ECG findings *Ant view* silhouette now slightly enlarged, exceeding size of fist, pulsations normal, L 12.6 cm, B 10.1 cm Course: athletic activities discontinued Re-examination 6 weeks later *Ant view* L 12.2 cm, B 9.5 cm, practically identical with figures from first examination Example of influence of athletic activity on a heart which had been damaged by a preceding infection This fact was brought out by functional response only (dyspnea, cardiac enlargement).

at the end of the first or at the beginning of the second week, amounting to an increase of 10-22% for the cardiac area.

*Pneumonia.* No material is available which fulfills the criteria for exact comparison. Displacements of the cardiac shadow and indistinctness of the border in case of extensive infiltration complicate the evaluation It is stated that lobar pneumonia causes dilatation more often than does bronchopneumonia.

*Myocarditis.* Roentgenological studies of the purulent form are unknown.

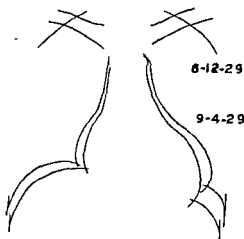


FIG. 131—38 yrs, M. Always well, Paroxysmal attacks of dyspnea for several weeks, lasting several hours. Moderate degree of cyanosis, dyspnea and liver enlargement. Marked perspiration. Cardiac enlargement, apical thrust widened, not resistant. Gallop rhythm. Rate 96, B P 105/70. Wassermann negative, E.C.G. n.s.r., a-v conduction time 0.22 sec, QRS of low voltage, notched, deep Q, T<sub>1</sub>, isoelectric, T<sub>2</sub> negative. Two roentgen ray studies with interval of three weeks. Diuretic treatment during this time caused 6 kg weight loss. *Ant view:* enlargement of silhouette without characteristic configuration.

L	B	T	Th
15.6	10.7	15.5	24.5
15.2	10.1	13.5	23.7

Pulsatory amplitude quite small at both examinations. Course severe failure following grippe infection. Sudden death. Postmortem. marked dilatation of rt. ventricle. Parietal endocarditis and extensive thrombosis in lt. ventricle. Microscopic findings: thrombi

phragm and heart became lower in position. T diminished proportionately more than the other two diameters. It is doubtful as to whether the heart size changed; resorption of some pericardial fluid might have occurred. The small pulsations, observed in the presence of cardiac enlargement of not excessive degree, expressed poor myocardial function.



FIG. 132—45 yrs, M. Always in good health, active in athletics; military service during war. Progressive dyspnea for 8 months. No pain, no palpitation. Pallor, slight degree of cyanosis, dyspnea at complete rest. Cardiac enlargement; apical thrust not palpable. Marked, diffuse tremor of chest wall over cardiac area. Permanent gallop rhythm. Rate 114. B P 110/65. Occasionally slight elevation in temperature, two chills during observation period of 7 weeks. E.C.G. n.s.r., initial deflection slurred, deep Q, R ax dev, T<sub>1</sub>, isoelectric. *Ant view:* elevation of diaphragm. Silhouette much enlarged, similar to shapeless lump of dough. Hardly any pulsatory amplitude. L 15.5, B 11.4, T 17.4, Th 27.5 cm. Postmortem. hypertrophy and dilatation of both ventricles, dilatation of rt. atrium. Parietal thrombi and remnants of parietal endocarditis in lt ventricle. No valvular disease. Opening of each coronary ostium normal in size. Descending branch of lt coronary artery moderately narrowed. Myocardium remarkable for its flabbiness, it almost flowed in the hand. Microscopic: no myocarditis, no appreciable degree of coronary sclerosis. Diffuse small scars in myocardium, more extensively in subendocardial layers. An instance of primary myocardial disease (toxic? infectious?) Predominance of transverse dimension which is either found with marked pericardial effusion or when cardiac dilatation is accompanied by a loss of muscular tonus. Roentgenological appearance of what has been described as "flabby heart".

Another type, interstitial, diffuse or focal, with but little or no parenchymatous necrosis, and without vascular endarteritis, is often overlooked in its milder forms. With or without known preceding infection, tachycardia, dyspnea and oppression, gallop rhythm and often abnormal electrocardiographic findings are present and last for perhaps a week or so. The roentgenologic findings may be negative in every respect. Other forms have a prolonged course with development of definite cardiac hypertrophy and dilatation, and show signs and symptoms of cardiac failure. Roentgenologically, one finds a globular enlargement of both sides with some straightening of the left border. The extent

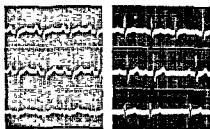
Prominence is noted of 1, lt atrium dorsad, and of 2, rt ventricle ventrad *Lt ant. obl view*: 3, lt bronchus elevated and narrowed, due to enlargement of lt atrium Note a) These roentgenograms were taken during a bout of sinus tachycardia with a high rate; b) here and subsequently, the target-film distance was 6 ft for the ant view and 4 ft. for the oblique views

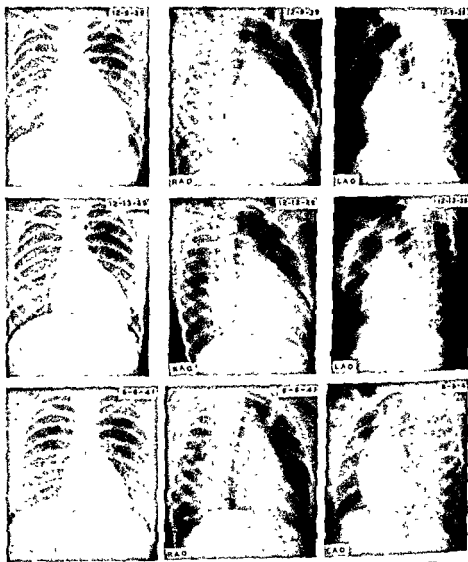
*Course*: Bed rest and 12 cat units of digitalis in the course of 3 days Findings Temp normal There was no cyanosis and the liver was normal in size The apical thrust was widened and heaving in the 5th isp 2 cm to the lt of the mid-clavicular line, with the precordial area no longer revealing a systolic forward thrust  $P_2$  was moderately accentuated, a very short systolic apical murmur was heard in the semilateral position following exercise The rhythm was regular, the rate 64/min BP 115/65 ECG. Nsr, P-R interval 0.16 sec, S-T<sub>1</sub> slightly depressed, T<sub>1,2</sub> positive

*Ant view*: The silhouette is still large but shows regression in size The air content and the vascular pattern of the lung fields is practically normal *Rt ant obl view* The dorsal and ventral prominence has receded *Lt ant obl view* The dorsal and ventral prominence has receded In particular the lumen of the lt bronchus is larger and its course steeper

*Course*: Circulation time for arm to tongue (saccharin) 13.5 sec, wbc 13750 with 59% polys, sedimentation rate 10 mm/1 hr Computed volume of heart, determined orthodiagraphically, 568 cm<sup>3</sup> For a period of four weeks the patient was kept on digitalis and strict bed rest The basic heart rate was 72 to 80/min, but at times there was sinus tachycardia with a rate up to 150/min The patient was permitted to be out of bed for one week. Re-examination (1-20-40) showed no evidence for infection nor for failure, there was no gallop rhythm and no murmur but the apical thrust had moved out 1½ cm. The ECG (18 cat units of digitalis during the preceding 16 days) showed nsr, rate 100/min, P 0.10 sec in width and notched, voltage of  $P_2$ , 2 mm, P-R interval 0.16 sec, electrical axis plus 60° (see illus) The computed volume of the heart was 685 cm<sup>3</sup> She was now kept on a maintenance dose of digitalis, was permitted to walk short distances, and to go to school Re-examination two months later revealed the findings essentially unchanged, she still showed a tendency to develop attacks of sinus tachycardia at times She had no complaints The wt was 45.0 kg (101 lb) The computed volume of the heart was 707 cm<sup>3</sup> She was permitted to go to school, abstaining from physical exertion

For more than a year she took daily 0.1 gm (grs lss) of digitalis and 0.6 gm (grs IX) of quinidine sulphate Findings (6-6-41, 1½ yrs following the first study) Wt 47.7 kg (105





FIGS. 135, 136, 137, 138, 139, 140, 141, 142, 143, 144—11 and 12½ yrs, F. History Negative for rheumatic fever and chorea. Two wks prior to the first examination, the patient had a sore throat, and experienced malaise, there was a temporary improvement; 3 days prior cough and hemoptysis occurred and several attacks of tachycardia were noted, of prolonged duration and associated with an almost imperceptible radial pulse. Findings: Rectal temp 37.7° C (100° F). There was a slight degree of cyanosis and pallor and, while the veins in the neck were not engorged, the liver was found to be slightly enlarged. There were a few rales along the base of the rt lung. The heart was markedly enlarged with widened and heaving apical thrust in the 6th isp in the ant axil line, and with the precordial area presenting a forward thrust during systole.  $P_2$  was accentuated, there were no murmurs. The rhythm was regular, the rate at times 130/min, at times 250/min. Rbc 4.8 mill, hb 12 gms, wbc 20500 with 75% polys. ECG: Basically sr, rate 130/min. P 0.12 sec in width and notched, P-R interval 0.16 sec, electrical axis plus 90°, T<sub>1</sub> of low voltage. Alternation with bouts of auricular tachycardia with a rate as high as 251/min.

*Ant view:* The silhouette is enlarged. The waistline is full, and a mitral configuration is noted. There is present a slight degree of pulmonary congestion. *Rt ant obli view:*

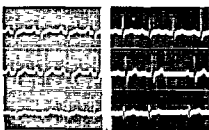
Another type, interstitial, diffuse or focal, with but little or no parenchymatous necrosis, and without vascular endarteritis, is often overlooked in its milder forms. With or without known preceding infection, tachycardia, dyspnea and oppression, gallop rhythm and often abnormal electrocardiographic findings are present and last for perhaps a week or so. The roentgenologic findings may be negative in every respect. Other forms have a prolonged course with development of definite cardiac hypertrophy and dilatation, and show signs and symptoms of cardiac failure. Roentgenologically, one finds a globular enlargement of both sides with some straightening of the left border. The extent

Prominence is noted of 1, lt atrium dorsad, and of 2, rt ventricle ventrad. *Lt ant. obl view*· 3, lt. bronchus elevated and narrowed, due to enlargement of lt atrium. Note: a) These roentgenograms were taken during a bout of sinus tachycardia with a high rate; b) here and subsequently, the target-film distance was 6 ft for the ant view and 4 ft for the oblique views.

*Course*: Bed rest and 12 cat units of digitalis in the course of 3 days. Findings: Temp normal. There was no cyanosis and the liver was normal in size. The apical thrust was widened and heaving in the 5th isp, 2 cm to the lt of the mid-clavicular line, with the precordial area no longer revealing a systolic forward thrust.  $P_2$  was moderately accentuated, a very short systolic apical murmur was heard in the semilateral position following exercise. The rhythm was regular, the rate 64/min. B.P. 115/65. ECG· N.s.r., P-R interval 0.16 sec, S-T, slightly depressed,  $T_{1,2,3}$  positive.

*Ant. view*· The silhouette is still large but shows regression in size. The air content and the vascular pattern of the lung fields is practically normal. *Rt ant obl view*· The dorsal and ventral prominence has receded. *Lt ant obl view*· The dorsal and ventral prominence has receded. In particular the lumen of the lt bronchus is larger and its course steeper.

*Course*· Circulation time for arm to tongue (saccharin) 13.5 sec, w.b.c. 13750 with 59% polys, sedimentation rate 10 mm/1 hr. Computed volume of heart, determined orthodiagraphically, 568 cm<sup>3</sup>. For a period of four weeks the patient was kept on digitalis and strict bed rest. The basic heart rate was 72 to 80/min, but at times there was sinus tachycardia with a rate up to 150/min. The patient was permitted to be out of bed for one week. Re-examination (1-20-40) showed no evidence for infection nor for failure, there was no gallop rhythm and no murmur but the apical thrust had moved out 1½ cm. The ECG (18 cat units of digitalis during the preceding 16 days) showed n.s.r., rate 100/min, P 0.10 sec. in width and notched, voltage of  $P_2$  2 mm, P-R interval 0.16 sec, electrical axis plus 60° (see illus.). The computed volume of the heart was 685 cm<sup>3</sup>. She was now kept on a maintenance dose of digitalis, was permitted to walk short distances, and to go to school. Re-examination two months later revealed the findings essentially unchanged, she still showed a tendency to develop attacks of sinus tachycardia at times. She had no complaints. The wt was 45.9 kg (101 lb). The computed volume of the heart was 707 cm<sup>3</sup>. She was permitted to go to school, abstaining from physical exertion.



first apical sound impure, with murmurs absent in any position, and also following exercise, B.P. 110/60. ECG (no medication for preceding 7 days): S.r. with sinus arrhythmia, av. rate 90/min. P 0.09 sec. in width, voltage of  $P_2$  ¾ to 1½ mm, P-R interval 0.14 sec, electrical axis plus 42° (see illus.)

(continued on p. 252)



of pulsations is markedly diminished. This type of myocarditis occurs in all age groups. If it is encountered in childhood, its roentgenological appearance in the prolonged, often lethal form, resembles the so-called idiopathic congenital hypertrophy of the heart (see this chapter) and *pericardial effusion*. A peculiar type of myocarditis often associated with mural thrombi has been described in conjunction with child bearing. No role is played by hypertension, toxemia of pregnancy, bacterial infection, or thyroid or coronary artery disease. Serious heart failure and cardiac enlargement tends to develop during the postpartum period. Embolization is a frequent event. Some cases go on to death. Others recover and then a regression in heart size is observed.

*Rheumatic Infection.* A focal, interstitial myocarditis is invariably found, and changes in the coronary arteries are often met with. Not all patients with active rheumatic carditis show cardiac enlargement. This is probably due to adequate management, which chiefly means reducing the load on the heart to a minimum by complete bed rest. When cardiac enlargement does result, it is

*Ant. and both oblique views.* As compared with the previous studies, a regression in heart size is noted, though a moderate degree of enlargement is still present. Computed volume of heart, determined orthodiagraphically, 524 cm<sup>3</sup>.

*Comment.* The regression in heart size is instructive with respect to both the 4-day interval at the beginning and the 1½-yr follow-up study. Detailed figures are as follows:

View	Diameter	Date		
Target-film distance		12-9-39	12-13-39	6-6-41
Ant } 6 ft }	Oblique	16.3 cm	15.5 cm	13.5 cm
	Broad	12.7	11.4	10.1
Rt. ant obl } 4 ft }	From conus to junction of inf vena cava with rt atrial border	12.6	10.8	9.5
Lt ant obl } 4 ft }	From cardio-hepatic angle to caudal border of lt bronchus	13.7	12.5	11.5

The patient probably had mitral stenosis and cardiac enlargement to begin with, and myocarditis plus rapid heart action brought on additional enlargement. Bed rest and medication with digitalis led to a reduction in heart size. There was apparently a transient increase in heart size again, judging by the orthodiagraphic volumetric determination, but as time went on a definite diminution took place, though a moderate degree of enlargement persisted. Note the following features: a) *Roentgenologic.* The regression in heart size seemed to affect all the cavities and, in particular, as visualized in the lt ant obl view, the course of the lt bronchus changed. b) *Clinical.* As the enlarged rt ventricle contracted, it took on a greater depth and, held ventrad by the enlarged lt atrium, it caused a systolic forward lift over the precordium. This sign disappeared as cardiac enlargement receded. c) *Electrocardiographic.* The change in the electrical axis indicated disappearance of cardiac strain of the rightsided type. And the return to normal of the P deflection pointed to a diminution in the size of the lt atrium and probably also to the disappearance of an interatrial conduction disturbance. Summarizing a), b) and c) A number of the findings in the beginning were compatible with the anatomic diagnosis of mitral stenosis except that the auscultatory evidence was lacking. Probably an instance of mute mitral stenosis.

primarily caused by the myocardial injury, and the most important contributing factor is physical exertion. Progressive cardiac enlargement means persistent active infection, and in children and adolescents congestive heart failure develops because of such infection. A marked degree of cardiac enlargement is of serious prognostic import. It has been claimed that the inactive rheumatic heart in the growing organism may decrease in size, in relation to the chest, until it becomes normal in size. This claim requires further study because a critical analysis of published material reveals first, that silhouettes were smaller but also the position of the diaphragm was lower; second, that the presence and subsequent resorption of pericardial fluid was not excluded with certainty.

The chief clinical findings in a typical instance of rheumatic pericarditis are, a massive dullness on percussion over the sternum that frequently extends rather far laterally in the second and third left interspaces; a friction rub; occasionally severe precordial pain that is relieved when the patient sits up or leans forward. Quite frequently the amount of exudate is small and no characteristic roentgen findings are visible, but occasionally a rapid accumulation of a considerable degree is observed. Resorption may also occur rapidly—even within a few days. It may so happen that digitalis is used simultaneously and the regression in the size of the silhouette may then be erroneously attributed to its pharmacological effect on the heart muscle. That the heart has actually receded in size can be ascertained roentgenologically perhaps only by demonstrating reduction in size of single cavities of the heart. This, however, may be expected only for cases that had previously shown an established valvular lesion. Active rheumatic myocarditis is likely to cause enlargement of those cavities that are already under considerable strain, such as the left ventricle in the presence of an aortic valvular lesion, or the left atrium and the right ventricle in the presence of a mitral lesion. These chambers may recede in size on subsidence of the acute rheumatic process. When the process becomes inactive, it will be seen that the already established alteration in the size and shape of the heart, due to a chronic valvular lesion; is constant. This rule of irreversibility does not hold for the recurrence and subsidence of rheumatic activity.

The early stage of a valvular lesion cannot be diagnosed by the roentgenologic study. Full-sized pulsations are noted in conjunction with an existing myocarditis. They are not specific in character and cannot be distinguished from those that are observed in the course of anemia, thyrotoxicosis or febrile diseases.

It cannot be emphasized too strongly that unnecessary heart enlargement may result when physical activity is resumed while the infectious process is still active or when the period of convalescence is too abbreviated. The advisability of resuming normal activity or continuing restricted activity rests with the functional response. For this, the size of the heart represents one of the objective criteria. Constancy of the heart size and shape permits a liberal attitude; progression in size and change in shape indicates the necessity of complete bed rest. The practical procedure is to make a standard roentgenogram of the chest during the earliest possible period of the disease. This pro-

cedure must be repeated at intervals of 2-3 weeks at first, and at intervals of 4-5 weeks for six months after the stage of convalescence is reached. A well established technique and the application of all the criteria for comparison are of fundamental importance. (Details have been given in chapter IV).

**NON-INFLAMMATORY CONDITIONS.** The most common etiology is represented by coronary artery disease. The association of coronary artery sclerosis

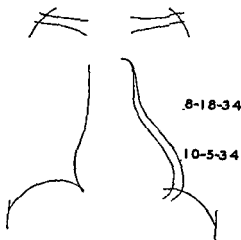


FIG 145—54 yrs, M Diabetes for 10 yrs Known to have had B.P 140/90 Somewhat exhausted for 2 days. No pain, no dyspnea Referred because of marked tachycardia. Absence of failure. Rate 190. B.P. 105/75. E.C.G.: paroxysmal auricular fibrillation; high take off leads I, II, low take off leads III, IV, Q<sub>i</sub> absent, T<sub>i</sub> positive Cardiac infarction with anterior seat *Ant. view*: normal findings as to size and shape, pulsations small (tachycardia) Course: recovery. Follow up study 6 weeks later: no signs of failure Rate 108. B.P 140/95 E.C.G.: n.s.r, findings of former anterior infarction. *Ant. view*: slight enlargement of silhouette to lt, 0.6 cm along cranial aspect of left ventricular contour, 1.0 cm near apical region, as compared with previous findings. Pulsations quite small

with other conditions, notably hypertension, which cause enlargement, makes it difficult to gauge the extent of its influence. However, by virtue of resulting chronic myocardial ischemia, coronary artery sclerosis is apparently the sole cause of cardiac enlargement in some cases. But many a patient, who subsequently will develop cardiac infarction, reveals a normal heart size. The enlargement usually is predominantly to the left side and an aortic configuration prevails with a plump left ventricular contour. The aortic shadow is rather prominent, corresponding to the age of the patient. There is no undue enlargement of the left atrium unless definite failure is present. The amplitude of pulsations is usually in inverse ratio to the size of the silhouette. This will be understood if we assume for the sake of simplicity, that the heart is a sphere; then its volume is  $\frac{4}{3} r^3 \pi$ ;  $r$  having a smaller and larger figure for two hearts, respectively, it follows that in order to reduce both spheres by approximately the same amount of  $\text{cm}^3$ . (systole) the  $r$  of the smaller sphere must be reduced more than the  $r$  of the larger. Some hearts are very large, even without complicating em-

physema or hypertension, though the possibility of a pre-existing hypertensive state should not be forgotten, and their silhouettes may resemble that of pericardial effusion. No definite correlation can be established between the size of the silhouette and the degree of disturbed function. The appearance of the lung fields, however, permits some conclusions as to the degree of left ventricular failure. When compensation is restored under the influence of digitalis and rest, an increase in the pulsations along the borders of the silhouette is noted, especially in the left lower pole region.

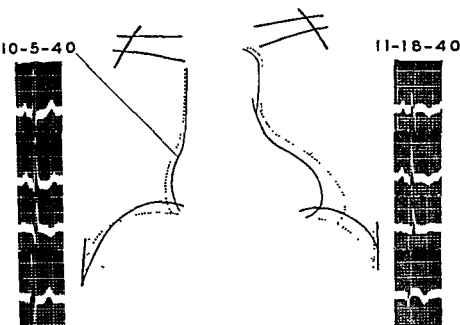


FIG 146—49 years, F History Weight loss and occasional palpitation Nothing pointed to cardiac infarction in the past, there was no effort limitation Hospitalized to standardize diabetic condition that was first noted two yrs prior Findings Ht 150 cm (4 ft 10 in), wt. 62.8 kg (138 lb). The heart was normal, BP 140/80 Fundi Normal Wbc 6650 Blood sugar 137 to 243 mgm %, Wassermann negative ECG S<sub>r</sub>, rate 96/min, normal P and P-R, Q<sub>s</sub> present, S-T segments slightly depressed in I, II, CF<sub>4</sub>, T<sub>s</sub> negative

Orthodiagram (10-5-40) Transverse silhouette of normal size Fluoroscopy showed normal pulsations

Course: On 10-11, the day she was to be discharged, she went into an attack of rage because she was to be demonstrated to students, and within one hr developed the clinical picture of cardiac infarction, verified electrocardiographically and followed by fever, leucocytosis and mild left ventricular failure A localized systolic pulsation could be palpated in the 3rd and 4th lt interspaces a few days later Findings (11-18) Wt 58.2 kg (128 lb) The abnormal pulsation persisted, there was no evidence for failure, BP. 115/70 ECG: S<sub>r</sub>, rate 132/min, QRS diminished in voltage, absence of the normal intrinsic deflection in CF<sub>4</sub>, S-T segments arched and T negative in I, II, CF<sub>4</sub>

Orthodiagram (11-18-40) The cardiac silhouette has increased in size, the oblique diameter is 14.5 cm as compared with 11.9 cm previously Fluoroscopy revealed no conclusive abnormality of pulsations but simultaneous palpation demonstrated the area of abnormal chest wall pulsations to coincide with the upper and middle portion of the left ventricular area

Course: Re-examination 15 mos. later showed all the objective cardiac findings unchanged BP 140/90 The patient had led a life without physical overexertion and had no complaints

Comment. Unquestionably this diabetic patient had degenerative heart disease to begin with The shape of the original ECG even raises the question of a minor posterior infarction in the past The shape and size of the heart were normal An anterior infarction developed and was soon followed by the clinical findings indicative of cardiac aneurysm The cardiac silhouette revealed an increase in size without any associated clinical evidence of failure One of the not infrequent instances where the clinical diagnostic method—palpation—is superior to the roentgenologic method

Preferably, one should not make a diagnosis of so-called idiopathic (congenital) cardiac hypertrophy. While it must be readily admitted that several cases on record seem to represent a kind of muscular gigantism, yet many such cases have been shown to reveal one of the following conditions: muscular degeneration and replacement fibrosis of infectious and toxic origin, coronary

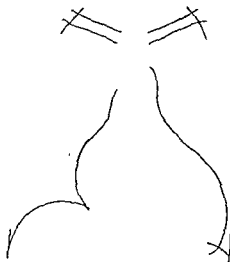


FIG 147.—42 yrs, M. Attacks of angina pectoris since age 36. Dyspnea since age 40. Apical thrust in axillary line, of increased resistance. Gallop rhythm. Soft apical systolic murmur. Heart sounds hardly audible, tachycardia, rate regular. B.P. 110/95.

of aortic shadow. Congestive lung failure. Lt. ant. obl. view (illustration omitted). Mass of heart bulges dorsad and caudad. Pulsations small. Sudden death. Postmortem: coronary sclerosis, myocardial fibrosis. Aneurysm of internal wall and apical portion of left ventricle. The wall was thinned out from within and it was not possible to make diagnosis of aneurysm from outer aspect of specimen only. An example of difficulty of roentgenological diagnosis of aneurysm of heart in the presence of a very large silhouette. Pulsations are commonly found to be small or even absent unless a marked degree of valvular regurgitation is present.

cy have been established. First, the incidence of subsequent attacks of cardiac infarction is somewhat higher in cases with enlargement than in those with a heart of normal size. Second, the probability that, following recovery from an attack of cardiac infarction, a patient will be able to lead a fairly

artery disease, abnormal origin of one or both coronary arteries, glycogen storage disease, diffuse rhabdomyomatosis. In addition, vitamin B<sub>1</sub> deficiency should be thought of. In all these instances, death usually occurs within the first two years of life preceded by signs and symptoms of cardiac failure. The roentgen ray appearance of the silhouette is that of a large, globular mass with predominant enlargement to the left. The silhouette may resemble that of a pericardial effusion.

**Cardiac Infarction and Cardiac Aneurysm.** **INFARCTION.** This is a relatively frequent event and commonly follows coronary artery thrombosis. The latter condition most frequently is an episode in the course of coronary atherosclerosis. So-called acute dilatation of the heart does not occur in conjunction with cardiac infarction. However, cardiac enlargement develops with the appearance of congestive heart failure, and a localized enlargement, a cardiac aneurysm, may soon make its appearance. The heart size may remain normal even following recurrent attacks of cardiac infarction. However, perhaps two-thirds of all the patients who have recovered from cardiac infarction do show enlargement of the heart, by far the most important cause being hypertension. Certain significant relations of heart size to functional capacity and life expectan-

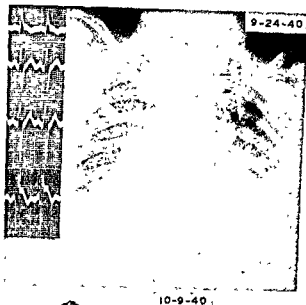
active life is almost twice as great in those with normal sized hearts as in those with cardiac enlargement. Third, the heart is increased in size in nearly all cases in which dyspnea is the sole cause of restricted activity. Fourth, the onset and incidence of angina pectoris subsequent to the attack of cardiac infarction is not related to the size of the heart. Fifth, congestive failure is found only in patients with enlarged hearts. And finally, sixth, patients with large hearts average almost as long lives as those with normal sized hearts.

The type of pulsations that are roentgenologically observed with cardiac infarction are of interest, but for the diagnosis of infarction—omitting here from consideration certain features in the history and clinical findings—the roentgenologic method has not the value of the electrocardiographic method. This holds true for all phases of the condition. But it is appropriate to point out that the E.C.G. may be influenced by digitalis medication or by association with an interventricular conduction disturbance, both of which complicate the diagnosis; that infarctions of multiple seat may cause difficulties in interpretation, and that, as time goes on, a previously abnormal tracing may return to a perfectly normal pattern. It is chiefly for these reasons that the roentgenologic study of pulsations is of value.

That pulsations corresponding to an infarcted muscle area would undergo alterations, might be postulated a priori. This has been substantiated by animal experimentation. Almost immediately following occlusion of branches of the coronary arteries in the dog, the region of the heart muscle involved ceases to contract, and a reversal of movement takes place during systole. The raised intraventricular pressure distends the ischemic area.

An opportunity to study acute cardiac infarction in man offers itself in those not too infrequent instances where a roentgenologic study is undertaken because of chest pain thought to be of other than cardiac origin, or where the particular diagnosis of infarction is not readily suspected because of a general cardiac picture. But if the diagnosis of acute cardiac infarction is once established, the patient must be saved from any effort and excitement and, therefore, must not be submitted to a roentgenologic examination.

When an attempt is made to diagnose local muscle changes by the study of pulsations, the following points deserve attention. First, absent or even reverse pulsations occur at the apical region when the heart is considerably enlarged. Second, in using a kymograph, its horizontal slits record only the horizontal component of motion and, since the crano-caudal movement may prevail in the apical area (and this certainly holds true for the vertical type of heart), the graph is likely to show a loss of excursion in this area—a loss that is only apparent. Third, observation is made difficult in the presence of a fairly large-sized extrapericardial triangular shadow. It therefore follows that the supra-apical area in a normal or only slightly enlarged heart affords the best chance for a diagnosis. Fourth, as compared with the basal portions of the left lower contour, the supra-apical area normally shows a lag in movement, and this finding must not be interpreted as abnormal. Fluoroscopic examination is best carried out in the anterior view with the patient holding his breath in deep inspiration. A rapid heart action is unsuitable for diagnosis, and a temporary slowing may be obtained by pressure on the carotid sinus. This should be ap-



FIGS 148, 149, 150—52 yrs., M. History: 5 yrs ago the patient had a kidney operation. During the past few mos he developed polydipsia, polyuria, and occasional dyspnea on exertion. He experienced tight retrosternal pain with radiation into the lt arm, lasting intermittently for a period of 4 days 3 wks later the pain returned, was more severe and constant in nature. After 3 days he sought medical advice, feeling faint. Findings: Pallor and slight cyanosis were noted. The heart was moderately enlarged to the lt, and an embryonic rhythm was heard. B P 148/100. The blood showed 260 mgm % of sugar,



21 mgm % of urea n.; the Wassermann test was negative, 18150 leucocytes, and a sedimentation rate of 19 mm/1 hr. The urine showed 5 gms of sugar in 100 cc, many leucocytes and a few rbc's. ECG: Nsr, P and P-R normal, initial deflection with  $Q_1$ , deep  $S_{1,2}$ , absence of intrinsic deflection in the chest leads, S-T interval elevated in lead I and the chest leads, depressed in leads II and III.

*Course:* A slight degree of fever lasted for 6 days. A transient friction rub was heard. A glucose tolerance test showed a typical diabetic curve. Insulin and diet were instituted. The sedimentation rate remained elevated. Serial ECG's showed appearance of a negative T in lead I and the chest leads, and bundle branch block at times. Clinically, the patient did well. The B P varied: systolic from 100 to 110 and diastolic from 70 to 80. In the fourth week following his admission, he developed an upper respiratory infection; low grade fever appeared; dyspnea, cyanosis, tachycardia, and pulmonary congestion were noted.

*Ant view:* The cardiac contour is prominent to the lt with visualization of the apical region obscured by some pleural fluid at the lt base. Pulmonary congestion is noted. Intravenous pyelography showed extensive pyelonephrosis of the rt. kidney.

*Course:* Digitalization was instituted and the patient's condition improved temporarily.

plied on one side only, the patient must be recumbent, and even then the procedure is not altogether safe for patients who have far advanced degenerative heart disease

A localized systolic expansion, which is a reversal of the normal, is a positive finding, and a localized diminution or absence of pulsation is a highly suggestive finding. Such findings, when obtained by fluoroscopy, are frequently indefinite, and graphic registration is therefore desirable. The extent of the infarcted area seems to be of greater import than its location. It would seem that even roentgenkymographic studies do not enable one to reliably locate the site of the infarct. This is perhaps partly a corollary to the fact that multiple infarcts are frequently found on postmortem study. Findings may be positive at first but may become non-conclusive subsequently.

**ANEURYSM** It occurs in about 9% of all cases of cardiac infarction studied at necropsy. By far the most common cause of such aneurysms is coronary occlusion. Location of this process in the anterior descending branch of the left coronary artery may lead to the formation of an aneurysm that is situated at the apex or at the ventral wall of the left ventricle, or which may involve both areas. Aneurysms of apical location, while of course supradiaphragmatic in nature, are buried in the left liver lobe. A large sized aneurysm of the ventral portion of the left ventricular wall, including the interventricular septum, may extend to the right and thus considerably displace the right ventricle to the right. The dorsal wall of the left ventricle will be infarcted when the circumflex branch of the left coronary artery is occluded, but aneurysm formation in this area is infrequent. Patients afflicted by cardiac aneurysm do not generally have a hypertensive background, and it seems that a ventricular wall thickened from hypertension is less prone to yield when infarcted than a heart with a nearly normal wall. An aneurysmal thinning may develop within a few weeks

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Low grade fever persisted. Suddenly there developed severe abdominal pain and distention, pulsations in the lower extremities diminished. Death within two days.

**Postmortem:** The heart was enlarged. There was a slight bulge noted in the supra-apical area with discoloration and softness on palpation (hardened specimen). The sup vena cava, rt ventricle, and lt atrium were found to be enlarged. The lt ventricle showed a thinning out of its wall over a large area, extending from the apical portion into the lower two-thirds of the interventricular septum. The wall measured 0.4 to 0.5 cm. thick, and showed marked fibrosis and a more recent infarction (opened specimen). The coronary arteries and their branches showed evidence of marked atherosclerosis. The descending branch of the lt coronary artery was completely occluded. The valvular orifices were normal. Microscopic: Fibrosis, healed infarction and recent infarction. Both lungs were congested, and there was a large area of a very recent infarction in the rt. lower lobe. There were about 100 cc of fluid in the lt pleural cavity. The rt kidney showed pyelonephrosis. The superior mesenteric artery was thrombosed, the tail of the thrombus protruding into the aortic lumen.

**Comment.** Cardiac infarction led to an aneurysmal thinning out of the left ventricular wall. Left ventricular failure was apparently precipitated by respiratory infection, and was accompanied by an hydrothorax limited to the lt side. Left ventricular failure associated with regular rhythm favors the development of a left sided effusion. The patient's grave condition did not warrant a fluoroscopic study which might have helped to establish the diagnosis of cardiac aneurysm. The electrocardiographic finding was highly suggestive. Pulmonary infarction and mesenteric artery thrombosis were the final events and the cause of death. Courtesy Dr L. A. Soloff, Temple University Hospital.



after cardiac infarction. With progressive healing, the affected portion of the ventricular wall becomes thinner and a bulge may or may not result. The thinning out process may manifest itself by a shallow pitting on the inner surface of the ventricle and will thus be perceived only upon inspection of the inside of the heart. The resistance of the fibrous and attenuated ventricular wall may be considerably fortified by the thickening of the epicardium resulting from an adherent pericardium and the deposit of layers of thrombotic clot



FIG 151—57 yrs, M Status anginosus 4 yrs previous and again a few months previous. Normal heart sounds. No abnormal pulsations. ECG: deep  $Q_2$ , deep  $T_1$ , (two infarcts with different site). Coronary artery disease, cardiac infarction. Ant view: aortic configuration, bulging prominence corresponding to cardiac aneurysm following infarction area of left ventricle (wh arrows). Courtesy Dr R. Huft and Dr W. Dressler, Vienna.

along the inner surface. Calcification occasionally occurs in the wall of aneurysms and in adjacent thrombi.

The most important and most frequent clinical sign of cardiac aneurysm involving the ventral portion of the heart is a localized area of pulsation, usually at or above the level of the left fifth rib, near the mid-clavicular line. The presence of a cardiac aneurysm does not exclude good effort capacity.

Concerning static aspects, it would seem that the percentage of cardiac aneurysms without significant roentgenologic changes is rather high. A very experienced author reported that in one-half of his cases, diagnosed on the basis of typical chest wall pulsations, history and ECG findings, nothing characteristic was revealed by the roentgen studies.

The diagnosis of an aneurysm is very difficult or impossible in two types of location. First, and this is common, when confined to the apical region. Here the aneurysm develops at the diaphragmatic and hepatic surface, and there is noted merely an elongation of the left lower pole area. Second, and this is

rare, when the aneurysm affects the right side of the left ventricle, including the greater part of the septum. Then there results an uncharacteristic enlargement to the right. Such a finding should arouse suspicion in the case of a patient without evidence for heart failure in the past or present but with a relevant history and positive ECG findings. A rounded and enlarged left ventricle in a patient known to have had coronary thrombosis, and for whom previous hypertension has been excluded, raises the question of the possibility of a cardiac aneurysm. The study of the vascular pedicle will be of help. In spite of a large-sized left ventricle, the pedicle is found to be slender. In contrast to this, when hypertension is the cause of left ventricular enlargement,



FIG 152—65 yrs, M Angina on effort one year Attack coronary thrombosis three months previous following which patient stayed in bed but two days Systolic lifting over anterior chest wall BP 120/50 ECG type T<sub>1</sub> cardiac infarction, deep Q<sub>1</sub> and Q<sub>2</sub>, sharply negative T<sub>1</sub> and T<sub>2</sub> Lt ant obl view (sl degree) bulging prominence along lt contour (bl arrows) Fluoroscopy reveals systolic laterally directed pulsations, corresponding in time to chest wall pulsations Cardiac aneurysm following infarction Courtesy Dr W Dressler, Vienna

one notes a broad vascular pedicle, which results from unfolding of the aorta. The chief positive finding refers to a change in the ventricular contour, namely the presence of a bulge. The shape of this prominence is oval and it is more curved than the rest of the heart border, but a more angular outline is occasionally observed. The contour is smooth and in no view can the mass be separated from the bulk of the cardiac shadow. The size varies from a small protuberance to a large mass that may extend as far as the left chest wall. An aneurysm of the ventral wall of the left ventricle will present itself in the anterior view as follows. When it involves the cranial or middle portion, a bulge, or more rarely a localized lump, is noted. When the caudal half is involved, this part of the heart shadow takes on a blunt and sometimes nearly rectangular appearance. A ventral location escapes detection from the front. Hence the im-

after cardiac infarction. With progressive healing, the affected portion of the ventricular wall becomes thinner and a bulge may or may not result. The thinning out process may manifest itself by a shallow pitting on the inner surface of the ventricle and will thus be perceived only upon inspection of the inside of the heart. The resistance of the fibrous and attenuated ventricular wall may be considerably fortified by the thickening of the epicardium resulting from an adherent pericardium and the deposit of layers of thrombotic clot



FIG 151—57 yrs, M Status anginosus 4 yrs previous and again a few months previous. Normal heart sounds. No abnormal pulsations. ECG: deep  $Q_2$ ; deep  $T_2$ ; (two infarcts with different site). Coronary artery disease, cardiac infarction. Ant view, aortic configuration, bulging prominence corresponding to cardiac aneurysm following infarction area of left ventricle (wh. arrows). Courtesy Dr R. Hilt and Dr W. Dressler, Vienna.

along the inner surface. Calcification occasionally occurs in the wall of aneurysms and in adjacent thrombi.

The most important and most frequent clinical sign of cardiac aneurysm involving the ventral portion of the heart is a localized area of pulsation, usually at or above the level of the left fifth rib, near the mid-clavicular line. The presence of a cardiac aneurysm does not exclude good effort capacity.

Concerning static aspects, it would seem that the percentage of cardiac aneurysms without significant roentgenologic changes is rather high. A very experienced author reported that in one-half of his cases, diagnosed on the basis of typical chest wall pulsations, history and E.C.G. findings, nothing characteristic was revealed by the roentgen studies.

The diagnosis of an aneurysm is very difficult or impossible in two types of location. First, and this is common, when confined to the apical region. Here the aneurysm develops at the diaphragmatic and hepatic surface, and there is noted merely an elongation of the left lower pole area. Second; and this is

portance of rotating the patient slowly into the right anterior oblique view. In this view the pouch projects ventrad so that its cranial margin forms a more or less abrupt ledge on the ventral contour of the heart. On examination in the left anterior oblique view the aneurysm, if large enough, will project as a dense shadow within the cardiac shadow proper. The caudal extent of an aneurysm can be studied to advantage by letting the patient have alternate draughts of sodium bicarbonate and tartaric acid in solution. An aneurysm of the dorsal-cranial wall of the left ventricle, if small, is invisible from the front. If the size is considerable, there is noted a bulge filling the waist-line but still separated by a notch of the left ventricular contour. In the left anterior oblique view the dorso-cranial portion of the silhouette bulges, and in both oblique views a barium-filled esophagus reveals a localized displacement so that occasionally even dysphagia has been noted. An indentation of the esophagus will also apparently follow if the ventral portion of the left ventricle is the seat of a rather large aneurysm. It is caused by mere displacement of the remainder of the left ventricular mass plus the left atrium. Follow-up studies on the size and shape of cardiac aneurysms have revealed interesting observations. No change may take place over a period of years, or the bulge may progress in size; or the original pouch-like prominence may gradually smooth out and fuse more or less with the rest of the cardiac contour. Differential diagnosis of cardiac aneurysm should take into consideration an aneurysm of the sinus of Valsalva, of intrapericardial seat, that may project from the cranial portion of the heart in the region of the conus of the right ventricle. Calcification may involve the wall, the adjacent thrombi, or both. Its usual location is in the area of the left

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widened to 0.14 sec., notched, deep S in limb leads, intrinsic deflection absent in chest leads, S-T interval elevated in leads I, II and chest leads, depressed in lead III, T negative in leads I and lateral-dorsal chest leads, positive in leads II, III and ventral chest leads (Illustration A shows *orthodiagram* indicating points where ECG's were obtained by means of the exploring chest electrode and where the mechanical movements were registered)

*Ant view (B).* The pulmonary vascular markings are slightly accentuated, and at the lt side an area of fibrosis is noted. Both the great vessels and the heart are slightly shifted to the rt. There is a large bulge extending to the lt and caudad from the heart; here encircling lime salt deposits are noted (bl arrow). Fluoroscopy revealed faint pulsations along this bulge. *Rt ant obl view (C).* The bulge extends ventrad, and lime salt deposits are again noted (wh arrow). The esophagus deviates dorsad. *Lt ant obl view (D).* The heart is slightly shifted ventrad while the bulge extends far dorsad. In both oblique views the mass is seen to bulge into the stomach air bubble.

*Comment.* A large cardiac aneurysm developed following infarction of the heart. The effort capacity of the patient was reasonably satisfactory. The diagnosis is based on the history, and on clinical, electrocardiographic and roentgenological findings. With respect to the latter, attention is directed to the unusually large size of the aneurysm and to calcification in its wall. There is no other etiology to account for "left ventricular enlargement." The discrepancy between the size of the left ventricle and the rather slender vascular pedicle makes improbable a previous hypertension, which condition is commonly associated with a broad vascular pedicle due to unfolding of the aorta. Note the abrupt ledge along the cranial margin of the cardiac contour which is particularly evident in the rt. ant. obl view. Mild dysphagia should be accounted for by the displacement and narrowing of the esophagus. There is no evidence for constrictive pericardial disease which, although it may be associated with calcification, could not account for the huge size of the silhouette.



FIGS 153, 154, 155, 156—62 yrs, M History 9 yrs ago the patient developed retro-sternal burning, profuse perspiration, and was gravely ill. Subsequently he resumed and continued activity with moderation. On walking quickly, or with emotional disturbances, the anginal syndrome appeared, and occasionally a mild degree of paroxysmal nocturnal dyspnea occurred. There was never evidence for congestive failure. The chief complaints referred to insomnia and peculiar paresthesias in the extremities. There was a mild degree of swallowing difficulty. Studies 3 yrs prior revealed essentially the same findings as reported below. Findings Wt 57.3 kg (126 lb). The respiratory rate was 32/min, a few rales were noted at the rt base, and the venous pressure was somewhat increased. The caudal-dorsal-lateral portions of the lt lung revealed some dullness to percussion, and the breath sounds were diminished. The cardiac dullness extended from the lower part of the sternum into the axilla. Systolic pulsations were noted, extending from the cranial border of the lt 5th rib halfway between nipple and lt parasternal line to the lt 8th rib in the midaxillary line. Over this area the two heart sounds were of low intensity but there was no gallop. The rate was 90/min with an occasional ea. B.P. 110/76. Fundi showed nasal arterioles narrowed without evidence of sclerosis. Blood chemistry normal. ECG (no digitalis medication), Sr with occasional premature auricular and ventricular beats, P 0.10 sec in width, P-R normal, initial deflection

has been noted. Death ultimately resulted because of the malignant character of these tumors.

Differentiation from pericardial tumors and diverticulae and from certain cystic mediastinal tumors situated in the immediate vicinity of the cardiovascular silhouette might require a diagnostic pneumopericardium and pneumothorax. Most of these localized bulgings which have been verified were located on the right side.

Hydatid cysts which can extensively involve the heart and the cardio-



Figs 157, 158—85 yrs, M Always well until recent development of gastric malignancy Good cardiac function, no murmurs, BP 120/80 ECG normal *Ant view* elongation of aorta Normal appearance of cardiac silhouette Sickle-like area of calcification is noted (bl arrow); fluoroscopy reveals marked movement *Rt ant obl view* lime salt deposits clearly demonstrated (bl arrow) Calcification in the fibrous ring at the base of the mitral valves, discovered in the course of routine study of the chest

pericardiovascular surface have been reported by roentgen studies, they cause little interference, although as soon as they burst, sudden death occurs as a result of embolization

**Cardiac Calcification.** The amount of the calcareous deposits in the heart is relatively small and conditions of contrast are not favorable Full adaptation of the eye and the use of a small shutter in fluoroscopy is of paramount importance A small grid Potter-Bucky diaphragm may be used to great advantage. The roentgenographical demonstration requires the use of a fine focal spot, shortest possible exposures, and some over exposure The method of choice is the use of a Rotalix tube with a small cone, at a target-film distance of 90-120 cm Extrinsic areas of calcification such as costal cartilages, bronchi and glands are excluded by adequate rotation in fluoroscopy and by films which are either taken from several angles or by stereoscopic roentgenography.

**Mitral and Aortic Ring** The skeleton of the heart is placed between atria and ventricles. It consists of the membranous septum, fibrous trigona and fibrous annuli. The fibrous annuli for both the aortic and mitral valves

lower pole, and its appearance is cup-shaped. Occasionally a good part of the bulging left lower contour is encircled by a thin layer of calcium deposits. Calcification of the pericardium never affects this area exclusively. As a matter of fact, in the presence of pericardial calcification it is not uncommon to see the apical region free when there are extensive calcareous deposits around the rest of the heart.

The study of pulsations along an aneurysm reveals no uniform behavior. They are either absent or indefinite. They may not follow in orderly sequence. Or they are not synchronous with the pulsations of the adjacent heart contour. A paradoxical movement is not infrequently noted, namely, outward during systole; and, correspondingly, an appreciable thrust on palpation.

**GLYCOGEN STORAGE DISEASE.** This condition is known only in infants. The abnormal deposition occurs especially in the parenchymatous organs. Predominant or exclusive enlargement of the liver and kidneys represents one type. Another is characterized by an enormous enlargement of the heart affecting both the muscle mass and the size of the cavities. Extensive deposition of glycogen in the heart muscle may, however, exist without enlargement. Certain cases which have been published under the title of idiopathic congenital hypertrophy undoubtedly belong in this group of glycogenosis. The few available roentgen ray studies reveal a large globular silhouette.

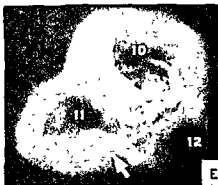
**Cardiac Tumors.** The clinical picture produced by tumors of the heart varies enormously, depending upon size, location, etc. Certain roentgenological findings, taken with the clinical picture, may be considered as indirect signs. Thus, a tumor or tumor-like thrombus of the left atrium may induce a marked degree of functional mitral stenosis and lead to the development of a silhouette which in all views is typical of a mitral heart. As another example, the vascular shadow appears markedly enlarged to the right when a sarcoma developing in the right atrium leads to a great enlargement and secondary thrombosis in the superior vena cava. Localized bulkiness of the silhouette is not common. Such a localized protrusion, however, was observed in one instance near the base of the heart and to the left, together with the clinical finding of a systolic impulse. Postmortem examination revealed a cavitated gumma which communicated with the left ventricular cavity. Malignant tumors which reach the epicardial surface are soon followed by pericardial effusion; other tumors increase in a diffuse manner (multicentric) and lead to an enlargement of the silhouette which is not characteristic. This holds true for the epicardial as well as for the myocardial growth. The latter type is represented by the diffuse rhabdomyosarcoma. An enlargement of the silhouette, with visible pulmonary metastases, should make one consider a metastatic tumor in the heart and/or pericardial space. The intradistinction of localized

out-pocketings of the silhouette, smoothly outlined due to the intrapericardial site; they are homogeneous in nature. Only one type of cardiac tumor shows expansile pulsation—the angiosarcoma. Deep roentgen ray treatment has been carried out in cases suspected as cardiac and/or pericardial tumors. In cases where the tumor was radiosensitive, a regression in the size of the silhouette

undergo dystrophic changes in a certain number of aging hearts, passing from fatty degeneration through necrosis and atheroma to calcification, which may reach such a degree as to produce disturbances in function, causing mitral stenosis and/or regurgitation and aortic stenosis. The calcareous deposits often extend far into the ventricular musculature and may reach the size of the little finger. The degree of associated coronary artery disease is not greater than one would expect to encounter in a group of patients of the same age without a marked macroscopic degree of annular and valvular calcification. Calcification is also sometimes observed as a late result of syphilitic and rheumatic valvular disease, though usually sparing the roots of the valves.

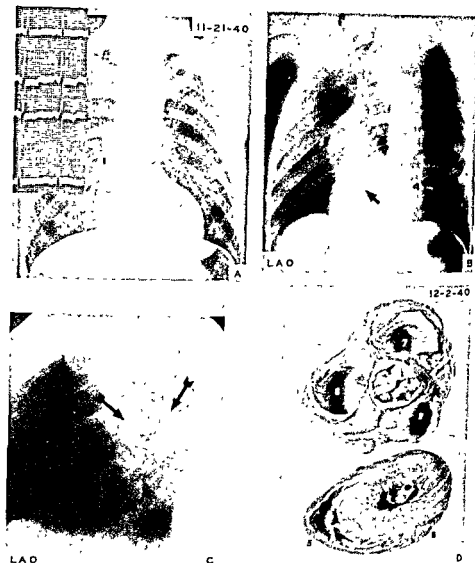
The approximate position of the different valve areas has been discussed in chapter III. Some change in their topographical relationship takes place necessarily in the presence of marked cardiac hypertrophy and dilatation. Suffice it to say here that in the anterior view the mitral valves are caudad and slightly to the left, and in the left anterior oblique view they are caudad and

*Postmortem:* The weight of the heart was 475 gms. The left ventricle was hypertrophied. The thickness of the lt ventricular wall was 3.0 cm and that of the rt ventricular wall 0.6 cm. The cavity of the lt ventricle measured 3.5 cm in its widest diameter, and the lumen of the rt ventricular cavity was greatly narrowed. The aortic cusps were deformed, thickened, and showed calcification which extended toward the mitral leaflets but did not involve them. There was an advanced degree of stenosis of the aortic orifice. The ascending aorta was dilated and bulged ventrad. In the region of the bulge the circumference of the aorta was 10.4 cm, and above this the vessel narrowed down to 8.5 cm. The remainder of the aorta showed numerous atherosclerotic plaques but there was no ulceration nor calcification. The coronary arteries showed very little evidence of atherosclerosis. Microscopic. Heart muscle, patchy myosclerosis, ascending portion of aorta, moderate degree of atherosclerosis and presence of a marked amount of elastic tissue, occupying practically the entire medial coat. In the stomach there was a large penetrating ulcer near the pyloric ring with erosion of an artery on its base and a massive hemorrhage. *Hardened specimen (D)*, lower half showing caudal aspect of heart, upper half showing cranial aspect of base of heart with orifices: 5, rt ventricle; 6, lt ventricle; 7, mitral orifice; 8, tricuspid orifice; 9, pulmonic orifice, center, deformed and calcified aortic cusps with gravely stenosed orifice. *Roentgenogram of base of heart (E)*: 10, mitral orifice, 11, tricuspid orifice, 12, pulmonic orifice, center, calcific deposits about aortic ring and cusps, lumen of rt coronary artery (wh arrow).



*Comment:* The high grade aortic stenosis was well tolerated, and the patient's death was not related to it. Roentgenologically, there was noted: 1. Evidence of lt ventricular hypertrophy, without an appreciable degree of dilatation. 2. Prominence of the ascending aorta. This was more marked than one would expect for this body build and age, and it was not due to disease of the wall. As a matter of fact the amount of elastic tissue was great. This patient was probably for many years the bearer of a dynamically dilated aorta, in connection with the valvular lesion, and this dilatation became finally fixed anatomically. 3. Calcification of the aortic orifice.





FIGS. 159, 160, 161, 162, 163—63 yrs, M History Operation for gastric ulcer 7 yrs previously, recurrent abdominal pain recently No dyspnea, no dizzy spells Findings. Slender build Emphysema was noted but there was no evidence for failure Left ventricular hypertrophy was palpated but a thrill could not be felt over the aorta A systolic murmur heard over the aorta was transmitted to the neck and to the apical area,  $A_2$  was almost inaudible BP 90/80 There was a mild hypochromic anemia, Wassermann negative, gastric analysis grade III hyperacidity and hypomotility Fundi Normal. ECG N s r., rate 82/min, left axis dev and slurring, S-T depressed and T negative-to-diphasic in limb leads, T isoelectric in CF. Fluoroscopy and roentgenograms of stomach showed large ulcer crater.

*Ant view (A)* Moderate degree of emphysema Aortic configuration of silhouette with elongation of aorta. 1, prominence of ascending aorta; 2, lt. ventricle with rounded-off contour *Lt ant obl view (B)*: 3, prominence of ascending aorta, 4, lt. ventricle, overlapping the spine; calcification of aortic ring and cusp area (bl arrow). *Lt. ant, obl view, coned down, higher penetration (C)*. Calcification of aortic ring and cusp area (bl arrows)

*Course.* Death following two severe gastric hemorrhages

of the heart in man, and of lipiodol deposits and metallic bodies introduced around and in the atrioventricular groove in animals, serve to prove the fallacy of cardiac output determinations which have for their basis the difference in the size of the cardiac silhouette in systole and diastole. A study of the locations of these deposits also has an important anatomical bearing because it shows the position of the mitral and aortic valves in the living. Judging from the available material, it seems permissible to state that these valves are farther to the right, i.e., nearer to the midline, in the living than one might expect from the anatomical textbooks

*Coronary Arteries.* Arteriosclerotic calcification of the media of these vessels



FIG 165.—Mitral valvular disease, predominant stenosis. Roentgenologic, volumetric reconstruction. Ant view: pear shaped appearance with enlargement to rt and lt cranial. Cranial shift of intersection between rt vascular and cardiac border (rt arrow), caused by increase of heart mass in height. Prominence of conus arteriosus (lt lower arrow). Distal portion of arch not well marked (lt upper arrow). Lt lat view, enlargement lt atrium (arrow). Courtesy Prof Dr. G. G. Palmieri, Bologna, Italy

exclusively, in contrast to the peripheral vascular system, is extremely rare. The atherosclerotic process may be in the nature of a calcifying stenosing type or of a clinically benign form with rigid walls and a wide lumen.

A fairly large amount of calcareous material is necessary before roentgenologic visualization becomes possible. The degree of calcification is no measure for a stenosing process. Calcification of the left coronary artery is more frequent and the chance to detect it is greatest in the left anterior oblique view because the anterior descending branch is then much foreshortened and any dense deposits will thus be intensified by projection. Calcific deposits reveal movements as follows: the anterior descending branch of the left coronary artery moves cranial and caudal when studied in the right anterior oblique view while for the left anterior oblique view a jerky movement and elliptical course is noted, the longitudinal axis of which extends from cranial-ventral to

definitely to the left of the aortic valves. Though the visibility in the latter view is not altogether good, yet differentiation of the two calcified valve or valve ring areas is satisfactory. The mitral valve area should be looked for toward the dorsal one-third of the cardiac shadow while the aortic valve area is likely to be found in the middle one-third. In the anterior view, these calcareous deposits may be expected near the midline, sometimes a few cm. to the left of it, at about the middle or within the lower third of the mass of the cardiac shadow. The projection of choice, for the beginning of the examination is a slight degree of right anterior oblique position ( $10-20^\circ$ ). Calcareous deposits in the fibrous rings are usually large and dense, sometimes sickle-like in appearance.

Fluoroscopy is indispensable in the diagnosis because the characteristic finding of these deposits is motion. A very small shutter must be used. These



RAO

FIG 164—65 yrs, F Angina pectoris since age 57, worse during last six months. Cardiac findings grossly negative. B.P. 180/88. Moderate peripheral sclerosis. ECG essentially normal. Rt ant obl view. marked calcification in descending branch of lt. coronary artery (short bl arrows). Courtesy Dr. Merril C. Sosman, Peter Bent Brigham Hospital, Boston.

movements are quite extensive; as much as 2 cm. in length, far greater than is noted at the peripheral borders of the heart. They are dancing or jerking in character, the speed of movement is not constant but rather sudden and steplike. The direction is toward the left lower pole during systole, the type an elliptic rotation. Intrinsic movements of the valves are occasionally noted and if both aortic and mitral valves are calcified they may move independently. In those cases in which a bandlike calcification is seen, the length will vary considerably in different positions but in all of them these deposits will appear within the cardiac shadow. The finding inside the heart shadow of a calcific shadow having the form and characteristic movements described above leaves scarcely any other diagnostic possibility than that of valve ring or valve calcification. Calcified thrombi present rather large, round or oval masses. Extrinsic areas of calcification may be projected outside of the heart shadow by rotating the patient.

The study of the movements of the calcified heart skeleton and valves, has an important physiological aspect. The large amplitude of these pulsations gives proof, or rather one of the proofs, that the pump-like action of the atrio-ventricular septum is of prime importance in the mechanics of cardiac contraction, and consequently for the discharge of blood. These findings, together with the roentgenological observations of bullets which have lodged near the base

is represented by metastatic calcification in osteitis fibrosa cystica, or by septic, toxic and pyemic conditions, all with renal involvement. A second group is accounted for by wall endocarditis. Coronary artery disease seems to be an occasional factor, although perhaps only a contributory one, in producing a rather diffuse calcifying degeneration. Calcification occurs in the wall of cardiac aneurysms and has already been dealt with. The calcifying process in constrictive adhesive pericardial disease often extends into the cardiac musculature but there is no way to differentiate such cases roentgenologically from others in which the process is limited to the pericardial layers. Sharply defined, round or oval, *irregularly calcified areas in the region of the atria may represent old organized thrombi.*

**Acquired Valvular Lesions.** These lead to characteristic changes, the degree of which depends upon several factors: degree and duration of the lesion; presence or absence of associated disease either in the heart muscle itself or in the peripheral circulation, physical demands on and constitutional type of the bearer. A more exact determination of heart size in the presence of a rheumatic valvular lesion is significant for prognosis. That life expectancy becomes shorter as cardiac enlargement increases, is shown by the following study. By the use of the cardiothoracic ratio the size of the heart was evaluated in 1164 patients afflicted with various types of rheumatic valvular lesions. The mortality quota was determined by follow-up studies. The ratio of actual to expected deaths for three groups with normal, slightly-to-moderately enlarged, and much enlarged hearts was 420%, 589% and 1092%, respectively.

**Mitral. ANATOMICAL FINDINGS** Quite frequently a combination of stenosis and regurgitation is found. Isolated regurgitation is quite rare in the adult who is afflicted by an old rheumatic valvular lesion. The ordinary combination of stenosis and regurgitation results in varying degrees of hypertrophy and dilatation of the right ventricle and left atrium and, eventually, of the right atrium. With mitral stenosis, the left ventricle is unchanged in size, or is quite small, and only an inconspicuous portion is visible from in front. The right ventricle expands far to the left since its outflow tract lies to the left and the left ventricle offers little resistance. Mitral regurgitation causes enlargement of the left ventricle; this prevents a marked expansion of the right ventricle to the left, hence the latter is forced for the most part to enlarge cranially. Thus the conus region is prominent in this condition also. The enlargement of the right ventricle produces a counter-clockwise rotation of the mass of the heart and great vessels, when visualized from the base. This is less pronounced with associated left ventricular enlargement. As a result of this rotation, parts of the right ventricle, especially the conus arteriosus, participate in the formation of the left border, the aortic loop holds a more sagittal course and the left lower pole region is displaced dorsad. Clinically the region of the apical thrust is likely to show a systolic depression. The mass of the right ventricle is found to be predominantly to the left of the midline; its development in the direction of the long axis of the body increases the height of the heart and its development in the anteroposterior direction leads to a broad contact with the anterior chest wall, resulting in increased flatness to percussion and a forward lift on inspection and palpation. A ventral displacement of the aorta and pulmonary artery is also present.

caudal-dorsal. The right coronary artery is best observed in the left anterior oblique view and shows chiefly a cranial and caudal movement, which is side-wise to the course of the vessel. The following technical conditions will give optimal results in roentgenography: 40 k.w. rotating anode tube, 70 cm. target-film distance, 400 ma. and up to 85 k.v. Speed films reveal linear and segmental shadows, sometimes double and parallel, arranged in a flattened arc. Differentiation from calcification of the mitral ring and mitral and aortic valves is not difficult. The superficial site, the different structure and the minimal movement are sufficiently characteristic. Differentiation from peri-



FIG 166—18 yrs, M Chorea and rheumatic fever at age 12. Repeated attacks paroxysmal dyspnea and bloody expectoration since age 16. Palpitation. Systolic propulsion of precordium and retraction of apical region. Presystolic and systolic thrill and murmur at apex  $P_2$ +++, BP 90/78 ECG - n.s.r. Mitral stenosis with attacks of severe cardiac asthma. Ant. view. aortic knob small. Pulmonary conus prominent (bl arrow). Cardiac shadow much higher than vascular shadow. Intrapulmonary vascular shadows increased in size and number, causing in part a fine mottled appearance of lung fields. Lumina of some bronchi visible (short wh. arrows).

cardial calcification located along the coronary sulcus is more confusing and must be based upon a different structural appearance: more solid in the case of pericardial disease. Extrinsic areas of calcification must be excluded by fluoroscopy and by films taken from different angles or by stereoscopic roentgenography.

Aneurysms rarely occur and they probably come in conjunction with a congenital structural anomaly plus calcific degeneration. One such case has been observed which revealed during life a shallow outpouching near the base; postmortem verification was obtained.

**Muscle and Endocardium.** The number of cases in this group is small and if one excludes calcification in cardiac aneurysms and thrombi, no roentgenologic intravital diagnosis has apparently been made so far. One group

The left atrium enlarges in whatever direction space is available: towards the spine, diaphragm, bifurcation of the trachea and structures of the root of the right lung. A considerable enlargement towards the left side is unusual; the auricular appendage may or may not participate in the formation of the left heart border. The expansion of the left atrium induces varying degrees of the following displacements of the adjacent structures: right ventricle and right atrium ventrad; aorta and pulmonary artery ventrad and craniad; bifurcation of the trachea and left bronchus dorsad and craniad; esophagus dorsad and to the right. Occasionally, however, the esophagus is displaced to the left; fibrous adhesions may account for this. When this occurs the average angle of the tracheal bifurcation is  $95^\circ$  (minimal  $78^\circ$ , maximal  $117^\circ$ ), the normal figures are  $69^\circ$ ,  $58^\circ$  and  $78^\circ$ , respectively. The lumen of the left bronchus and of the esophagus may be considerably narrowed. Atelectasis of the base of the left lung may result, for instance, in the course of bronchitic mucous membrane swelling and this has sometimes been erroneously interpreted as pneumonic consolidation. Rarely is the swallowing function interfered with. The structures of the right hilus are displaced craniad and to the right in those instances in which the atrium expands far toward the right side. Excessive degrees of enlargement always bear to the right, are in no relationship to the type of the lesion of the mitral orifice and represent a true aneurysm forma-

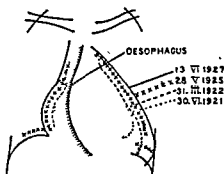


FIG 171—33 yrs, F Rheumatic history Deliveries 5 and 3 years previous Cardiovascular failure for six months Mitral stenosis and regurgitation, inactive. Rate regular *Ant view* progression of size of silhouette over course of 6 years is noted, with increasing mitralization of shape and lowering of diaphragm Esophagus displaced to rt and dorsad.

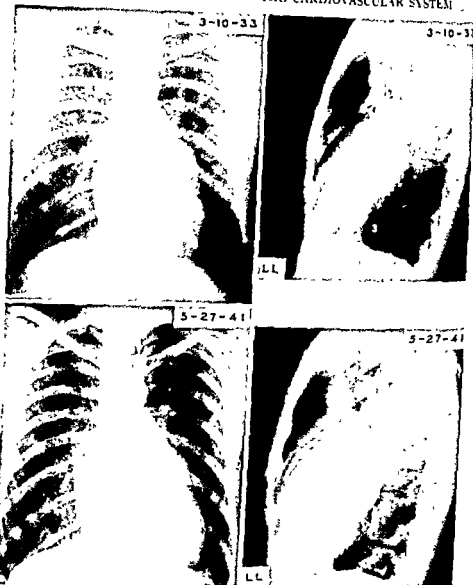
	L	B	T
1921	12.3	8.8	12.1
1922	14.6	10.1	13.6
1925	15.5	11.8	15.0
1927	19.3	14.0	17.4

Postmortem mitral stenosis and regurgitation Marked hypertrophy and dilatation of both ventricles and both atria

(1941—8 yrs following first study) Wt 74.1 kg (163 lb) Heart somewhat larger than previously, otherwise no changes ECG Auricular fibrillation

*Ant view.* The silhouette has increased, and the configuration is unchanged The lt atrium is just about visible at the rt cardiac border (5) *Lt lat view.* There is a marked prominence of the rt ventricle ventrad and craniad, and of the lt atrium dorsad It is furthermore noted that the width and depth of the chest have increased, as compared with the findings 8 yrs ago

*Chest.* Depth increased 1.5 cm



FIGS 167, 168, 169, 170—24 and 32 yrs. M History Rheumatic infection at the age of 9 yrs Complaints referred to dyspnea on effort and an occasional hemoptysis. Findings (1933) Wt 75.4 kg (166 lb) There was no evidence for failure There was a systolic precordial forward lift, and marked pulsations were noted in the 3rd and 4th interspaces The findings were those of mitral stenosis with a slight degree of mitral, and perhaps also aortic, regurgitation BP 120/70 ECG NsT, P widened to 0.13 sec and notched, rt axis dev.

*Ant view* The diaphragm is low, and the vascular pulmonary markings are increased The cardiac silhouette is enlarged, with the conus of the rt ventricle unduly prominent (1) *Lt lat. view* The rt ventricle is prominent ventrad (2), and, dorsally, the Lt ventricle (3) appears normal There is a moderate prominence of the Lt atrium (4) dorsally Note: These and the following roentgenograms were taken at a 4 ft target-film distance The rt ant. obl view showed the conus prominence ventrad

*Course:* 4 yrs later (1937) auricular fibrillation became established There were mild rheumatic pains at times The effort capacity decreased somewhat The patient led a quiet life and took digitalis regularly At no time did he develop congestive failure Findings

chapter X). The external outline of the pulmonary artery and of the dilated conus arteriosus form a diagonal which merges caudally into the more or less steep line of the left heart border. The lumen of the aorta tends to be narrow in those cases in which the mitral lesion was acquired during childhood or adolescence and where there is predominance of stenosis. As previously mentioned the course of the aortic arch is almost sagittal.

**ROENTGENOLOGICAL FINDINGS. STATIC ASPECTS. Anterior View.** With acquired mitral lesions the aortic knob is small or entirely hidden within the shadow of the spine. The second arch joins the aortic knob high cranially and appears, provided that the aortic knob is not visible, as a first arch. It is more or less prominent and may be distinguished from another convex arch situated further caudad, i.e., the conus, or both arches may blend. The waist of the heart appears filled out or, to express it differently, the distance of the silhouette from the midline, at this level, is considerably increased above normal findings; the normal concavity is often replaced by a convexity. Because of the considerable length of this middle arc, a lowering of the intersection with the left lower contour results. The latter may be steep and but little curved and the distance of its farthest point to the left of the midline is well within normal limits; this finding is characteristically noted in the presence of a high degree of mitral stenosis, unless a very marked increase in the size of the right ventricle causes an increase in the development to the left. In the presence of well marked regurgitation one finds the contour longer, more curved, and that the distance of its farthest point to the left of the midline is increased above normal figures. The right side of the vascular contour is straight in outline and relatively short. The right cardiac contour is longer than normal, markedly curved and the distance of its farthest point to the midline is considerable. The silhouette in mitral disease thus varies between the form of a vertically placed egg and a triangularly shaped

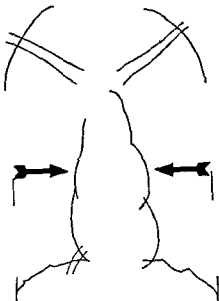
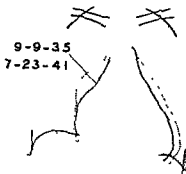


FIG 174—31 yrs, F Dyspnea, cough and palpitation for years Asthenic type, small linear chest Height 175 cm, weight 44 kg Mitral stenosis without cardiac enlargement. ECG n.s.r., right axis dev., moderate degree Ant view silhouette vertical. Upper third: small vascular shadow with moderate prominence of pulmonic arch Middle third: contours of silhouette prominent on either side, corresponding to lt atrium (bl arrows) T at this level 7.7 cm Lower third: area not enlarged L 10.7, B 8.5, T 8.9 cm Fluoroscopy mesial movement of middle lt. contour precedes mesial movement of lower lt. contour. Hilar vessels slightly enlarged Low position of diaphragm with basal adhesions Obl views (illustrations omitted) narrowing of retrocardiac area at level of lt atrium, no undue prominence ventrally An example of the effect of mitral



tion, the wall being extensively fibrosed and the content in exceptional cases amounting to as much as 2 liters of blood. Chest wall pulsations and auricular fibrillation are concomitant clinical findings, and atelectasis at the right base and a depression of the diaphragm and the liver surface are noted anatomically.

The part of the heart that becomes prominent between the pulmonary artery and the left ventricle is either the conus of the right ventricle or the



FIGS 172, 173—42 and 38 yrs. M. History: Effort limitation had started 7 yrs previously. Findings (6 yrs previously): A mild degree of congestive failure was noted. The heart was very large, a precordial forward lift, a double mitral valvular lesion, auricular fibrillation, and a normal blood pressure were noted.

*Orthodiagram (9-19-35)* Large heart showing a typical mitral configuration and a marked prominence of the left atrium posteriorly.

*Course.* The patient rested most of the time and took digitalis regularly. Fatigue and dyspnea were always present and there was mild congestive failure at times. Findings essentially the same as previously except that the heart had further enlarged.

*Orthodiagram (7-23-41)* The size of the silhouette is further increased and the left atrium now bulges into the right lung field. *Roentgenogram:* Lipiodol was injected through a catheter passed into the trachea in order to outline the trachea and the main bronchi. One notes the widened angle of the bifurcation of the trachea, and the elevation of the bronchi, particularly of the left one (bl arrow).

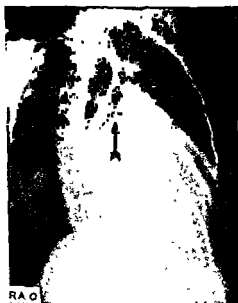
*Comment:* A demonstration of the influence of left atrial enlargement, on the angle of the tracheal bifurcation and on the position of the main bronchi.

enlarged left auricular appendage, or the prominence may be composed of both of these. The more marked the conus enlargement and the rotation of the heart to the left, the less will the left auricular appendage participate in the formation of the border, it may still encircle the conus, or it may stay behind it. Low position of the diaphragm tends to rotate the heart to the right, hence the conus becomes less prominent and the left auricular appendage more so.

The lumen of the pulmonary artery is found to be slightly or moderately enlarged, and the vessel is displaced cranial, ventrad and to the left (see also



Figs 177, 178, 179—28 yrs, F Chorea at age 7 and 8, valvular lesion found at age 18. Rheumatic fever at age 25. Large heart. Mitral stenosis and regurgitation, aortic regurgitation. B.P. 90/60. E.C.G. auricular fibrillation. Ant view large silhouette, mitral configuration. Barium filled esophagus visualized to deviate to rt (bl arrow). Aortic knob visible (upper wh arrow), prominence of lt atrium into lt lung field (lower wh arrow). Rt ant obl view mass of heart protrudes ventrad and dorsad, displacement of esophagus. Dark oval area noted at root of lt atrium (short bl arrow), corresponding to lt bronchus which is elevated so much that its course is practically identical with direction of projection. Lt ant obl view large lt atrium projects far dorsad (lower wh arrow). Lt bronchus noted to be elevated (bl arrow). Aortic arch visible (upper wh arrow). Unusual development of lt atrium far to left. Courtesy Dr George W. Holmes, Mass Gen'l Hosp, Boston.



the enlarged left atrium in connection with the total increase in the depth of the heart, and is influenced by the sterno-vertebral diameter of the chest. With minor degrees of enlargement of the left atrium the course of the esophagus shows no deviation from the midline, but with moderate or excessive distention there is invariably deviation. This displacement, as mentioned above, usually is to the right, rarely to the left. With minor degrees of displacement, it superimposes or just surpasses the border of the spine, with greater

Adequate technique permits visualization of a denser area within the silhouette, usually best seen in the area to the right of the spine; this additional shadow is produced by the left atrium. Thus in certain instances a double instead of a single right cardiac contour is noted; the intersection of the upper arch is easily followed as it curves caudally and mesially. The upper contour corresponds to the left atrium, the lower to the right atrium and their different depths can be easily demonstrated as follows: First, by a horizontal shift of the roentgen ray tube, the degree of excursion for the two respective contours is different, greater for the cranial, smaller for the caudal contour (parallax); second, by slow rotation into the two anterior oblique views; it is noted that the cranial shadow corresponds to a mass farther dorsad whereas the caudal



FIGS 175, 176—31 yrs, F Rheumatic fever in childhood Palpitation and moderate dyspnea on exertion since age 16 Never had signs of congestive failure Marked cardiac enlargement with dullness in the rt intrascapular space and marked transmission of systolic murmur along spine ECG—auricular fibrillation Mitral stenosis and regurgitation, inactive Ant view: the 2 films were taken at an interval of 3 yrs. Typical mitral configuration (a) L 18, B 12 1 (measured to prominence at lt contour), T 16 8, Th 21 8 cm Central pulmonary vessels enlarged Double contour at rt The upper one corresponds to an aneurysmal enlargement of lt atrium (wh arrow), the lower one to contour of rt atrium (bl arrow) (b) L 19 1, B 13 2 (measured to prominence at lt contour), T 17 9 cm Th unchanged. The rt cardiac contour is now entirely formed by aneurysmatically dilated lt atrium

shadow is farther ventrad. Finally, the entire right cardiac contour may be formed exclusively by the left atrium (aneurysm) approaching or blending with the right chest wall shadow; the right atrial border is then noted to be mesial, within the total cardiac shadow. The right hilus structures are elevated, and the medial aspect of the diaphragmatic contour depressed. It follows that the right cardiac border can be formed either by the right or, in these rare instances, by the left atrium. And while in the former case a marked dilatation to the right is always associated with clinical signs and symptoms of right-sided failure (distension of the right ventricle or of the right atrium or of both), the latter finding is compatible with a fairly good compensation since the other three chambers need not reveal simultaneously a high degree of enlargement.

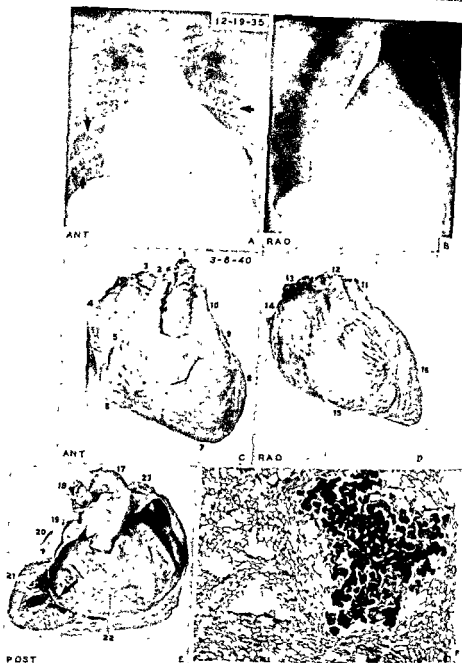
The degree of displacement of the esophagus is determined by the size of



FIGS 177, 178, 179—28 yrs, F Chorea at age 7 and 8, valvular lesion found at age 18. Rheumatic fever at age 23. Large heart. Mitral stenosis and regurgitation, aortic regurgitation. B.P. 90/60. ECG: auricular fibrillation. Ant view large silhouette, mitral configuration. Barium filled esophagus visualized to deviate to rt. (bl arrow). Aortic knob visible (upper wh arrow); prominence of lt atrium into lt lung field (lower wh arrow). Rt ant obl view mass of heart protrudes ventrad and dorsad, displacement of esophagus. Dark oval area noted at roof of lt atrium (short bl arrow), corresponding to lt bronchus which is elevated so much that its course is practically identical with direction of projection. Lt ant obl view large lt atrium projects far dorsad (lower wh arrow). Lt bronchus noted to be elevated (bl arrow). Aortic arch visible (upper wh arrow). Unusual development of lt atrium far to left. Courtesy Dr George W. Holmes, Mass Genl Hosp., Boston.



the enlarged left atrium in connection with the total increase in the depth of the heart, and is influenced by the sterno-vertebral diameter of the chest. With minor degrees of enlargement of the left atrium the course of the esophagus shows no deviation from the midline, but with moderate or excessive distention there is invariably deviation. This displacement, as mentioned above, usually is to the right, rarely to the left. With minor degrees of displacement, it superimposes or just surpasses the border of the spine, with greater



FIGS 180, 181, 182, 183, 184, 185.—39 and 44 yrs, M History First attack of irregular heart action 8 yrs. prior to first examination, subsequently becoming permanent Occasional hemoptysis Pat working but easily fatigued Findings (age 39): Ht 168 cm (5 ft. 6 in), wt. 61.4 kg (135 lb) A slight degree of cyanosis and dyspnea on effort, but not at rest, were noted The lungs revealed harsh basal breath sounds and a few rales The heart showed a precordial bulge and a systolic forward lift, with the apical thrust in the 6th lsp mid-axill line, heaving and resistant; systolic soft tissue depression in 5th lsp; and at the apex a diastolic thrill and rumble,  $P_2$  was accentuated B P. 120/90 Rhythm irregu-

degrees it projects a definite distance from it. A comparatively small lateral shift is compatible with a marked dorsal displacement; the lateral shift predominates in other instances. The lumen above the cranial turning point may be widened, thus representing a prestenotic dilatation. When the size of the lumen at the segment of deviation is studied in different views, a proper opinion of the shape of the lumen of the deviated portion of the esophagus may be obtained by a study in two views that are at right angles to each other.

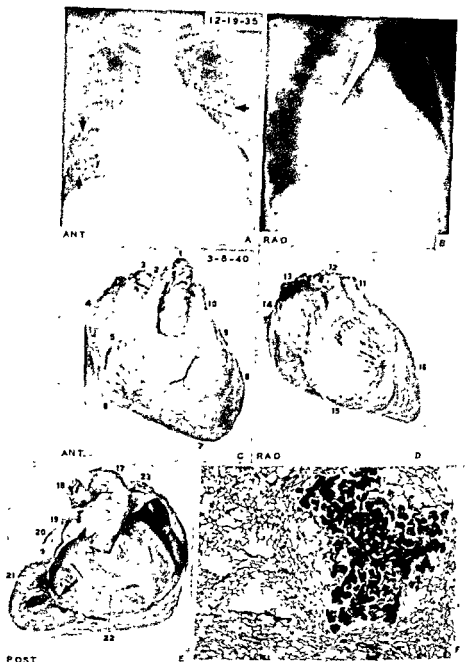
lar, vr 66, pr 50 ECG. Auricular fibrillation, no axis dev., digitalis effect. Other laboratory findings essentially normal.

*Ant view (A)* Small calcific nodules are distributed throughout the lung fields (arrows). The vascular pattern is but moderately increased. The silhouette is large and has a mitral configuration (orthodiagraphic oblique diameter 17.8 cm). The rt. border bulges considerably to the rt and also cranial, and is formed by the very large left atrium. *Rt. obl view (B)*: The rt. ventricle bulges ventrally and the lt atrium dorsally; the esophagus is much displaced.

*Course* The patient rested much, took digitals, and for 5 yrs his condition changed very little. There followed an attack of precordial distress, wheezing and coughing. The local M.D. stated: "Heart ok, you have asthma"; two hypo's (? adrenalin) gave no relief. Three wks later a similar attack; 1 wk later hemoptysis, and the week following that an attack of sharp pain in lt. side of the chest was experienced. Findings (age 44). Wt 53.7 kg (120 lb). Veins in neck full, liver slightly enlarged, fine moist basal rales. Orthodiagram. The prominence of the rt heart border, i.e., of the lt atrium, has increased. Sudden death 4 days later.

*Postmortem*: The heart was excessively large and, emptied of blood, weighed 750 gms. The lt. border extended to the posterior axillary line. The ventricles occupied a transverse position in the chest, the rt. ventricle being found to the left of a line drawn through the lt sterno-clavicular junction, and thus was situated far to the left. The rt atrium was much dilated and thin walled. The lt atrium was very much enlarged and extended 4 cm to the rt of the rt atrial border. The marked enlargement of the lt atrium had the following effects: a) it caused a mild indentation on the surface of the rt. liver lobe, b) it pushed the rt atrium ventrad, c) it pushed the rt main branch of the pulmonary artery and the aortic arch cranial and dorsad, d) it caused both venae cavae to slope obliquely dorsad, and e) it widened the angle of the bifurcation and displaced the esophagus dorsad and to the rt. Dense fibrous tissue bridged the gap between the lt. and rt atria. The epi- and myocardium of the lt. atrium could be easily stripped off, leaving the thickened and fibrous endocardium. The mitral valve leaflets were thickened and calcified, and the mitral orifice admitted only the tip of the little finger. *Microscopic*: The wall of the lt. atrium showed grade 4 of myocardial degeneration with interstitial myocarditis and old pericarditis, probably rheumatic in origin. Great vessels: The aorta was small while the pulmonary artery and its branches were about normal. Lungs: Recent infarct in rt lower lobe, numerous small, calcified shotty nodules could be palpated throughout. *Microscopic*: The nodules were made up of adult bone containing marrow spaces and even a suggestion of marrow cells—multiple osteomata. *Hardened specimen, ant view (C)*: 1, arch of aorta; 2, sup vena cava; 3, rt main branch of pulm artery; 4, aneurysmatically enlarged lt. atrium; 5, fibrous tissue bridging gap between lt and rt atrium; 6, rt atrium; 7, rt ventricle; 8, lt ventricle; 9, lt auricular appendage; 10, pulm artery. Note the very large lt atrium displaces the rt ventricle to the lt and the rt main pulmonary artery branch cranial. *Rt ant obl view (D)*: 11, ascending aorta; 12, sup vena cava; 13, branches of rt main pulm artery; 14, aneurysmatically enlarged lt atrium, the epi- and myocardium partly stripped off; 15, rt atrium; 16, rt ventricle. Note the displacement of the rt.

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FIGS 180, 18  
heart action  
hemoptysis

wt 61.4 kg (135 lb). A slight degree of cyanosis and dyspnea on effort, but not at rest, were noted. The lungs revealed harsh basal breath sounds and a few rales. The heart showed a precordial bulge and a systolic forward lift, with the apical thrust in the 6th isp mid-axill line, heaving and resistant; systolic soft tissue depression in 5th isp; and at the apex a diastolic thrill and rumble;  $P_2$  was accentuated B P. 120/90 Rhythm irregu-

piration is not uncommonly seen and this phase of respiration should be avoided in taking films or evaluating the position of the esophagus during fluoroscopy. The bend is likewise fairly well marked in healthy children.

When the left bronchus has become sufficiently elevated, its lumen may be visualized as a disc-like transparent area just above the roof of the left atrium.

The aortic shadow is short. There is a considerable prominence noted in the region of the conus, and the mass of the right ventricle bulges in front. This leads, as the lateral views reveal, to a broad contact with the inner aspect of the anterior chest wall.

*Left Anterior Oblique View.* The aortic window is considerably narrowed. The main development of the mass of the heart is ventrad and toward the left upper quadrant. The junction of the cardiac and vascular contour in front is further craniad than normal, while dorsally one cannot always determine very distinctly the intersection between the left atrial and ventricular borders on account of interference with the shadow of the spine, other vascular structures and the diminished transparency of the lung fields. The left ventricular contour is apt to project, at the average middle degree of rotation, into or beyond the spinal shadow. This is either an expression of enlargement of this cavity or of mere displacement dorsad. The contour of the aortic arch is visualized poorly or not at all.

A deviation of the esophagus dorsad is not noted except in those instances in which the anterior view reveals the left sided type of displacement. The lumen of the left bronchus is often found to be displaced in the cranial direction and a marked degree of narrowing can be present.

This description of the typical changes will be followed by a short discussion of some special features.

The volumetric reconstruction of hearts with mitral valvular lesion (predominance of stenosis) shows the left lower pole directed dorsad and craniad and this dorsal displacement may be so marked that the clinical apical thrust must obviously be caused in these instances by the right ventricle.

The roentgenological findings pertain to the chronic stage of the disease. They are occasionally of decisive diagnostic value for the clinician, especially in the presence of rapid heart action and auricular fibrillation and also in instances of hemoptysis. Clinically thyrotoxic hearts may easily imitate the findings of mitral stenosis (see chapter VII). An early stage of the disease may well be associated with normal roentgen ray findings and an answer to the clinical question as to whether one is dealing with an organic or functional murmur is then often impossible. We do not know the average length of the interval between the onset of the endo-myocarditic rheumatic lesion and the appearance of changes in the silhouette. The amount of bed rest and physical activity seem to be among the influencing factors.

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for right ventricular enlargement is absent. No direct relation exists between the size of the left atrium and the degree of pulmonary overfilling. As a matter



There is seen a narrowing in one plane, while in the other the size is normal or even slightly increased, and the esophagus assumes the shape of a scabbard. An adequate technique permits of visualization of the widened angle of the tracheal bifurcation (this has also been demonstrated by injection of lipiodol), and, in certain instances, the narrowed lumen of the left bronchus.

*Right Anterior Oblique View.* A few cases have been observed in which a prominence at the level of the left atrium was lacking in spite of the clinical finding of long standing mitral valvular disease and corresponding characteristic roentgen ray appearance in the anterior view. The only anatomical explanation which can be offered is that the presence of dense mediastinal adhesions obscures an enlargement. A balloon-like prominence which completely or partly obliterates the transparent retrocardiac space, is a typical finding. In the great majority of instances a triangular, transparent space remains visible above the diaphragmatic contour and is noted to increase in size during inspiration. The retrocardiac space is not only completely overshadowed in the presence of an aneurysmal dilatation but, in addition, the mass of the left atrium considerably overlaps the vertebral shadow. The esophagus is displaced dorsad, with the cranial turning point rather abrupt.

A dorsal shallow bulge of the esophagus, without an abrupt change in the direction of its cranial segment, is noted when the depth of the heart is increased in the course of marked right ventricular dilatation and also when the base of the left atrium is displaced dorsad and cranial due to marked left ventricular dilatation. Care should be exercised to avoid diagnosing a definite enlargement of the left atrium in these instances; though it must be admitted that it is not possible either to prove or disprove the presence of a minor degree of enlargement. A slight dorsad curving of the esophagus during ex-

*Comment.* 1) The radiologic and anatomic features of an excessive enlargement of the lt atrium have been demonstrated. The radiologic silhouette in such cases may resemble that seen in the presence of pericardial effusion. 2) The functional status of the heart was rather satisfactory for a number of years in spite of such a large silhouette. This is explained by the fact that the enlargement of the heart chiefly affected the atria, while the ventricles were not over-distended. The final downhill course was probably aided by the appearance of a pulmonary infarct. 3) Nodular bone formation in the lungs occurs in association with chronic mitral heart disease. Healed milary tuberculosis and multiple osteochondromata give similar radiologic features. 4) With respect to the place and character of the apical thrust, the following points deserve attention. The normal apical thrust is caused by the lt ventricle, the chief factors being the lever-like forward and cranial movement of the apical portion, the clock-wise rotation of the heart, and the increased consistency of the heart muscle. As long as the lt ventricle is not completely displaced dorsad, a normal circumscribed apical thrust may persist. In the presence of considerable enlargement of the rt ventricle, the ventral wall of the heart is mainly formed by it, and so is the apical thrust, the lt ventricle having been pushed away from the chest

of the enlarged rt. ventricle to the lt, whereby the thrust corresponds to the precordium but rather to the axillary portion. The displacement of the rt ventricle to the lt, by the excessively enlarged lt atrium, is aided by the absolute or relative smallness of the lt. ventricle.

increased rotation of the heart, perhaps in connection with a higher position of the diaphragm, or an actual increased dilatation of either the conus or pulmonary artery, or both, or perhaps a combination of all of these factors.

An inspiratory position of the rib cage and a rather low position of the diaphragm is commonly noted, especially in the presence of marked mitral stenosis. It is assumed that by this mechanism the resistance to the blood flow through the capillaries of the lungs is kept as low as possible. Some degree of kyphosis is not rare, particularly in the presence of aneurysmal dilatation of the left atrium.

**DYNAMIC ASPECTS.** The caudal portion of the left lower cardiac contour

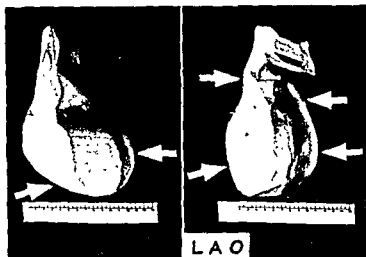


FIG 186—Young male. Rheumatic aortic regurgitation. Compensation. Roentgenologic, volumetric reconstruction. Ant view. moderate enlargement of lt. ventricle caudad and to lt (lt arrow) Rt ventricle (rt lower arrow) Lt ant obl view prominence of lt ventricle dorsad (lt lower arrow) Rt ventricle (rt lower arrow) Cranial portion ascending aorta (rt upper arrow) Lt atrium (lt upper arrow) Courtesy Prof Dr G G Palmieri, Bologna, Italy

often appears quite immobile in the anterior view when mitral stenosis predominates (rotation of the heart). Here the cardiogram of the apical region is likely to be normal. The pulsations are actually not absent; they take place mainly in the direction of the ray projection. This can be substantiated by turning the patient into the left anterior oblique view, the pulsations are then noted to extend farther and farther toward the lower pole region until a concentric movement is seen. A kymographic study shows a slow diastolic rise for these pulsations along the left lower contour. With marked predominance of mitral regurgitation, which causes little or no rotation at all of the heart, pulsations are noted along the whole left lower cardiac contour, and their amplitude is often increased in connection with the management of the additional amount of blood. The cardiogram of the apical region is positive. A kymographic study shows a rapid diastolic rise.

of fact, a very large left atrium is compatible with very little pulmonary overfilling; the large sac acts as a big pool for blood and thus takes off a load from the lesser circulation.

The appearance of the lung fields during the chronic stage of mitral disease has been referred to in chapter VI. Two further points deserve attention. Failure of the right heart with associated congestion in the greater circulation may, somewhat paradoxically, bring relief to the patient. Orthopnea and basal rales diminish Roentgenographically, the transparency of the lung fields increases, and the hilar shadows diminish in volume and increase in sharpness of outline. Physical exertion, the postpartum period or a febrile bronchitis favor the appearance of a transient pulmonary edema. Hazy and cloudy shadows of rather irregular distribution are then noted in the lung fields.

The inner topography of mitral hearts has been recently studied in the living by means of a contrast medium, with the resulting suggestion that the elevation of the left bronchus may be caused by the enlarged pulmonic veins rather than by the left atrium directly.

The changes in the silhouette which are noted in the course of a partial occlusion of the mitral orifice by a tumor are identical with those caused by mitral stenosis of common rheumatic etiology. Calcareous degeneration of the left fibrous annulus and of the mitral valves leads to the typical roentgen appearance of an ordinary mitral valvular affection. For the roentgenological diagnosis of these intracardiac calcareous deposits, see the preceding pages of this chapter. Thrombi cannot be visualized unless they contain areas of calcification.

*Differential Diagnosis.* A straight or convexly prominent middle portion of the left cardiac contour, often in association with a poorly visible aortic knob, is commonly noted in the presence of mitral valvular disease and hence the term "mitral-configuration." This term is purely descriptive and does not necessarily indicate that the mitral valves are affected. Such a (left) contour is seen physiologically in young individuals, especially of the hyposthenic type, and more often of the female sex, who often exhibit signs and symptoms of neurocirculatory asthenia. The presence of breathlessness and systolic (functional) murmurs may lead to the erroneous clinical diagnosis of mitral valvular disease. This configuration is likewise noted in the following conditions: emphysema, pneumoconiosis, a displacement due to a fibrotic process in the left upper lobe, thyrotoxicosis, beriberi, certain congenital cardiovascular malformations like patency of the ductus arteriosus, acquired disease of the pulmonary orifice and/or artery, right convex scoliosis of the spine leading to rotation of the heart, and sometimes in connection with adhesive mediastino-pericardial disease. The size of the left atrium, the appearance of the lung fields, and the pulsatory phenomena along the silhouette are of aid in differential diagnosis; further discussion is given in the respective chapters dealing with the aforementioned lesions. The clinical study is of first importance. Excessive enlargement of the left auricular appendage, of unknown etiology, is a very rare condition. The rest of the heart is normal.

The median convexity is often observed to be more prominent during the stage of heart failure. It is not known whether the cause for this finding is an

see-saw action results. The lateral movement of the cranial (left atrial) contour is simultaneous with the ventricular systole (this has been verified by kymography) and expresses the distension of the thin wall by regurgitating blood. This lateral movement is transmitted to the right hilus vessels, the pulsations of which should not be interpreted as intrinsic because no change in volume or density is noted and the phenomenon is limited to the right hilus only.

In some cases the left anterior oblique view permits one to observe pulsations along the caudal wall of the left main bronchus during ventricular systole, caused by the distention of the left atrium. This is also noted during bronchoscopy.

**Aortic. ANATOMICAL FINDINGS.** The etiologies are rheumatism, syphilis and atherosclerotic degeneration. The existence of functional regurgitation described by some in severe hypertension and anemia is doubted by others. A cystic degeneration of the media of the aorta may lead to a sufficient degree of dilatation of the aorta to cause functional regurgitation. Isolated stenosis of the aortic orifice is relatively rare and may be either acquired or congenital in origin.

Isolated aortic stenosis causes only moderate enlargement of the left ventricle but a marked dilatation is noted in the presence of aortic regurgitation. In certain instances, however, dilatation may be absent, viz., with associated constrictive pericardial disease, in individuals with very poor general muscular development (asthenic type), and when only a short time has elapsed since the development of valvular regurgitation. Dilatation affects especially the outflow tract so that the anterior wall of the left ventricle bulges considerably; the left ventricle is not demarcated by the anterior longitudinal sulcus but by a furrow a few cm. further mesiad which can be easily located by palpation, and originates where the thin, slightly depressed wall of the right ventricle meets the hypertrophied, bulging left ventricle. The interventricular septum bulges much farther to the right than the position of the interventricular sulcus would suggest. The main development is dorsad and here the left ventricle extends farther to the right than under normal conditions. Marked enlargement of the left ventricle is commonly noted with the rheumatic etiology and is easily compatible with good compensation, but failure is the rule in the presence of marked enlargement in association with syphilitic regurgitation, stenosis of one or both coronary artery openings accounts for this. The left atrium reveals hypertrophy but no dilatation, at least not during the phase of compensation.

The ascending portion of the aorta is commonly dilated when syphilis causes valvular regurgitation, excepting those cases in which the valves are affected very early by the supra-valvular site of the process. Both free aortic regurgitation and predominant stenosis of the aortic orifice, of rheumatic origin, may be associated with definite dilatation of the ascending aorta. This is referred to as dynamic dilatation although the exact cause is not fully determined as yet. Long standing dynamic dilatation may finally lead to a fixed dilatation, i.e., a dilatation that would also be present at postmortem investigation.

**ROENTGENOLOGICAL FINDINGS. STATIC ASPECTS. Anterior View.** In the

Pulsations at the level of the left auricular appendage alternate with those along the ventricular contour and are best noted in those instances in which first the waist of silhouette is more or less preserved, i.e., rotation of the heart and prominence of the conus arteriosus are not marked, and second where the volume and pressure changes in the left atrium are considerable as in mitral regurgitation. The pulsatory movements are less distinct or invisible in the

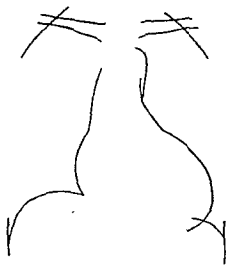


FIG 187.—34 yrs, M Rheumatic fever, pericarditis and subsequent aortic valvular lesion at age 20. Always physically fit No complaints Apical thrust heaving in 6th intercostal space, width of 2 fingers outside of midclavicular line Systolic depression of lower sternal portion Maximum diastolic murmur in 4th lt intercostal space. B.P. 135/25 E.C.G. nsr, lt axis dev Rheumatic aortic regurgitation, full compensation Ant view typical aortic configuration with enlargement of lt ventricle downward and to lt. L 15.4, B 10.8, T 14.8, Th 23 cm Pulsatory amplitude increased along all contours Lt. ant obl view (illustration omitted) lt ventricular area extends far dorsad, lt. atrium not enlarged These roentgenological findings were entirely unchanged over a period of 12 years.

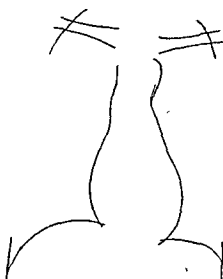


FIG. 188.—24 yrs, M Rheumatic fever and aortic valvular endocarditis at age 22 Asthenic type Heart not enlarged. Typical rheumatic aortic regurgitation Rate 76, B.P. 120/50 Ant view: silhouette of vertical type L 13, B 10.1, T 10.3, Th 24.3 cm Aortic knob slightly prominent. Low position of diaphragm Fluoroscopy reveals slightly increased pulsations at left lower pole Absence of cardiac enlargement and absence of "typical" configuration in the presence of an aortic regurgitation of 2 years duration The heart primarily was obviously of vertical, small type Exclusive use of T or of the T/Th ratio would lead to the erroneous assumption of an actually small heart. This error is prevented by using L and B

presence of auricular fibrillation. Other factors, as yet not well understood, seem to influence the visibility of these pulsations. In two instances of mitral regurgitation, a roentgencinematographic study revealed the following finding During early diastole, the left auricular appendage showed instead of the normal emptying a distension which lasted for  $\frac{1}{15}$  second.

When the extension of the left atrium into the right lung field partly overlaps the right atrial border, a peculiar appearance can be noted in a good many instances: the cranial and caudal contour alternate in movement so that a

tour projects into or, more often, far beyond the vertebral shadow.

The volumetric reconstruction of hearts with an aortic valvular lesion shows a tendency for the left lower pole to be directed slightly ventrad

An early stage of the disease may well be associated with normal roentgen ray findings and, as already stated for mitral valvular lesions, we do not know the average length of the interval between the onset of the valvular defect and the appearance of changes in the silhouette. With rheumatic etiology the age at which the lesion is acquired and the amount of bed rest or degree of physical activity seem to be but two of several influencing factors.

Calcareous degeneration of the aortic ring and the aortic valves should be looked for and anticipated especially when an aortic valvular lesion is present in an individual, though the roentgenological diagnosis of these calcareous deposits see the preceding pages of this chapter.

*Differential Diagnosis.* Some prominence of the aortic shadow to the right and left, and a prominence of the left lower contour associated with a deepening of the cardiac notch, are suggestive of aortic valvular disease.

the aortic valves are affected. Such a configuration is seen among aged individuals usually in combination with hypertension, physiologically in the hypertrophic state with a cranial displacement of the diaphragm, and in many

the silhouette is of aid in differential diagnosis but clinical study is of primary importance.

As heart failure develops, an enlargement of the silhouette occurs to the right, to the left, and craniad, and the waist of the heart becomes less pronounced. This is spoken of as the "mitralization": the secondary enlargement of the right ventricle accounts for it. The prominence of the aortic knob persists. The area corresponding to the left atrium is noted, in the oblique views, to be definitely increased. The transparency of the lung fields decreases and the lung pattern increases, the hilar vessels scarcely ever reach the size which is noted in the presence of chronic mitral valvular disease

Experimentally produced aortic regurgitation in animals very early shows some enlargement of the silhouette which becomes more marked in the course of a few weeks.

**DYNAMIC ASPECTS** In the presence of isolated or predominant aortic valvular regurgitation the findings are as follows: The maximum of pulsations is noted in the lower pole region while their amplitude is less in the basal portions. The change from the systolic to the diastolic form takes place very rapidly and uniformly. The marked increase in the pulsations along the left ventricular contour is also easily observed in the left anterior and in the lateral views. The movements of the aortic shadow are exaggerated correspondingly

presence of aortic valvular regurgitation the aortic knob is usually prominent. The degree of prominence is in part determined by the phase of the cardiac cycle in which the observation is made. Farther caudad the left contour is rather concave in its upper portion and then swings into the elongated, elliptical, markedly convex contour of the left ventricle. Thus the waist of the heart is very much increased. An exception is noted in those instances in which

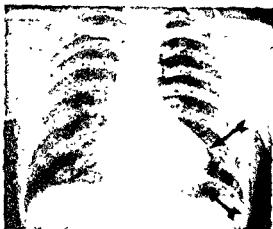


FIG 189—27 yrs, M Rheumatic fever at age 18. No complaints. Heart slightly enlarged, apical thrust slowly rising. Typical stenosis of aortic orifice without regurgitation. B.P. 100/85. Ant view. slight enlargement of silhouette. Lt lower contour globular in appearance, expressing marked hypertrophy with but slight dilatation (bl. arrows).

valvular regurgitation is superimposed on a heart which is in the vertical position at the start. The right side of the vascular contour is in its greater part convex in outline. Again, the degree of prominence depends upon the phase of the cardiac cycle. The intersection between vascular and cardiac contour is often lowered and the distance from the midline to the right contour is often moderately increased for both contours. Displacement accounts for it primarily. The shape of the silhouette in the presence of valvular regurgitation has thus been likened to a duck, boot, or horizontally placed egg. The transverse position of the heart is, however, more apparent than

real because of the fact that a considerable portion of the heart shadow penetrates deeply into the abdominal shadow. In the presence of stenosis of the aortic orifice, there is nothing unusual noted as to the aortic shadow; the left lower cardiac contour is rounded. An appreciable degree of enlargement is absent and when found the presence of an aortic valvular regurgitation or other

in, the ventricu-  
in increases, the

transparent retrocardiac space is seen in its entire length; its width is usually somewhat smaller in those instances in which the cardiac enlargement is considerable. A slight narrowing at the level of the left atrium may or may not be present. In the former case, one may be dealing with an actual enlargement of the left atrium or merely with a dorsal displacement. The esophagus is either not displaced at all or only to an insignificant degree. An abrupt dorsal deviation of the barium stream favors the diagnosis of left atrial enlargement.

*Left Anterior Oblique View.* The convexity of the aortic contour in front is moderately increased. The window of the aorta is preserved. The main development of the mass of the heart is dorsal and toward the left lower quadrant, and at the average middle degree of rotation the left ventricular con-

tour projects into or, more often, far beyond the vertebral shadow.

The volumetric reconstruction of hearts with an aortic valvular lesion shows a tendency for the left lower pole to be directed slightly ventrad.

An early stage of the disease may well be associated with normal roentgen ray findings and, as already stated for mitral valvular lesions, we do not know the average length of the interval between the onset of the valvular defect and the appearance of changes in the silhouette. With rheumatic etiology the age at which the lesion is acquired and the amount of bed rest or degree of physical activity seem to be but two of several influencing factors.

Calcereous degeneration of the aortic ring and the aortic valves should be carefully looked for and anticipated especially when an aortic valvular lesion of unexplained etiology is found in a middle aged or older individual, though it also occurs with the rheumatic and syphilitic types. For the roentgenological diagnosis of these calcareous deposits see the preceding pages of this chapter.

*Differential Diagnosis.* Some prominence of the aortic shadow to the right and left, and a prominence of the left lower contour associated with a deepening of the waist are commonly noted in the presence of aortic valvular disease and hence the term "aortic configuration." This term is purely descriptive as is also the term "mitral configuration," and does not necessarily indicate that the aortic valves are affected. Such a configuration is seen among aged individuals usually in combination with hypertension, physiologically in the hypertensive type with a prominent aortic knob, and in the atherosclerotic type with a prominent aortic knob. The importance.

As heart failure develops, an enlargement of the silhouette occurs to the right, to the left, and cranial, and the waist of the heart becomes less pronounced. This is spoken of as the "mitralization": the secondary enlargement of the right ventricle accounts for it. The prominence of the aortic knob persists. The area corresponding to the left atrium is noted, in the oblique views, to be definitely increased. The transparency of the lung fields decreases and the lung pattern increases; the hilar vessels scarcely ever reach the size which is noted in the presence of chronic mitral valvular disease.

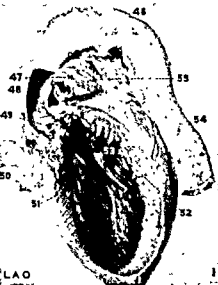
Experimentally produced aortic regurgitation in animals very early shows some enlargement of the silhouette which becomes more marked in the course of a few weeks.

**DYNAMIC ASPECTS.** In the presence of isolated or predominant aortic valvular regurgitation the findings are as follows: The maximum of pulsations is noted in the lower pole region while their amplitude is less in the basal portions. The change from the systolic to the diastolic form takes place very rapidly and uniformly. The marked increase in the pulsations along the left ventricular contour is also easily observed in the left anterior and in the lateral views. The movements of the aortic shadow are exaggerated correspondingly. It has been stated, as a result of kymographic studies, that the out-thrust begins almost simultaneously with the closure of the mitral valves, being more marked near the cardiac shadow; that there follows first a sudden retraction





FIGS 190-195



FIGS 190, 191, 192, 193, 194, 195, 196, 197, 198—54 and 57 yrs, M. History: Negative for rheumatism. Complaints referred to repeated spells of dizziness and slight dyspnea on more than usual effort. Findings (age 54) Ht 5 ft 6 in, wt 69 kg (152 lb). There was pallor but no evidence for failure. The apical thrust was heaving in the 5th isp in the ant axill line, a systolic thrill was felt over the aortic area, with a rough systolic murmur transmitted into the neck. A<sub>2</sub> was inaudible, and a very short diastolic whiff was heard. The radial pulse was rather small. Rate 78/min, rhythm regular. BP 120/75. ECG Nsr, normal P and P-R, initial deflection of increased voltage and width—0.12 sec, slurred, its axis dev, S-T segments depressed in leads I and II; T deep negative in I and II, positive in III. Blood Count normal, Wassermann negative. Fluoroscopy Large heart with aortic configuration, tortuosity of aorta,

calcified aortic valves visualized

*Course:* In addition to dizzy spells, there occurred a few syncopal attacks with generalized convulsions followed by slight coma. After 2 yrs the patient stopped working. He took digitalis only irregularly. One yr. later onset of congestive failure. Findings (age 57) Cheyne-Stokes respiration, pulmonary moist rales and systemic congestion were noted. The apical thrust was heaving in the 6th isp in the ant axill. line. On auscultation, findings were the same as previously. The radial pulse was small and of the plateau type. BP. 90/60, rhythm regular. Fundi negative.

*Ant view (A).* An aortic configuration with a marked degree of enlargement of the left ventricle to the left and caudad is noted, and the waistline of the silhouette is deepened. The aorta is normal in size. The lung pattern is nearly normal in appearance. *Rt. ant. obl view (B)* 1, trachea, 2, bifurcation of trachea, 3,3, dorsal and ventral aspects of tortuous

and then the true aortic wave, which shows a prominent incisura, corresponding to the second sound. Because of the wide excursions one is able to see the aortic arch in the oblique views in the young where under normal conditions fluoroscopy does not permit its visualization. It is observed in the anterior view that the amplitude of the right cardiac border is also increased. Further analysis reveals a pendulum movement for the whole heart shadow. A kymographic study shows that the lateral movement of the right cardiac border is simultaneous with that of the contour of the ascending aorta, precedes by a fraction

descending aorta, 4, rt. ventricle; 5, lt. ventricle; 6, conus of rt. ventricle, 7, bifurcation of pulm. art., 8, ascending aorta. *Lt. ant. obl. view (C)*: 9, trachea, 10, ascending aorta, 11, bifurcation of pulm. artery, 12, rt. atrium and ventricle; 13, lt. ventricle, enlarged; 14, lt. atrium, 15, lt. main bronchus; 16, tortuous descending aorta. Same view, *special technique (D)*: Calcareous deposits are noted (bl arrows), they correspond to the position of the aortic ring.

*Course*: Rapid recovery took place under morphine, digitalis, aminophyllin, and restriction of fluid and salt. One week later congestive failure recurred and was followed by an embolic occlusion of the lt. femoral artery. The rhythm remained regular all of the time. Cardiac asthma did not appear until the last day of life.

*Postmortem*: The weight of the unopened heart together with the large vessels was 1060 gms. The myocardium of the left ventricle measured 2.5 cm. in thickness, and the cavity was 4.5 cm. in diameter. Massive calcium deposits completely distorted the normal anatomy of the aortic valves, causing a very marked degree of stenosis, with the aortic orifice measuring at no place more than 0.2 cm. in width. The calcium deposits extended up the dorsal wall of the ascending aorta into the right atrium, and tended to partially occlude the ostia of the coronary arteries. The aorta itself appeared to be fairly free of atherosclerosis. There was patchy consolidation in the lt. lower lobe, a lt. sided purulent pleurisy and congestion in all the organs. Microscopic: The heart revealed rather diffuse fibrosis, many areas showed recent muscle fiber degeneration. *Hardened specimen, Ant. view (E)*: 17, arch of aorta, 18, sup. vena cava, 19, rt. atrium, 20, rt. ventricle, 21, sulcus corresponding to the interventricular septum, 22, lt. ventricle, 23, lt. auricular appendage; 24, pulm. artery, 25, descending thoracic aorta, 26, ligamentum arteriosum. *Rt. ant. obl. view (F)*: 27, arch of aorta, 28, ascending aorta, 29, sup. vena cava; 30, descending aorta; 31, rt. atrium, 32, rt. ventricle, 33, lt. ventricle, 34, conus of rt. ventricle, 35, pulm. artery. *Lt. ant. obl. view (G)*: 36, arch of aorta, 37, ligamentum arteriosum; 38, ascending aorta; 39, pulm. artery, 40, rt. auricular appendage, 41, rt. ventricle; 42, lt. ventricle, 43, descending thoracic aorta, tortuous, 44, lt. auricular appendage; 45, lt. branch of pulm. artery. Same view, roentgenogram, *Hardened specimen (H)*: The calcareous deposits at and around the aortic orifice are noted in the center of the picture. Using the terms "right" and "left" with respect to the specimen, we note: From the center itself arises the ascending aorta, coursing up and to the right, there follow the arch and the descending portion of the aorta. To the right and downward from the center, the right ventricular cavity is seen, from it ascends the pulmonary artery up and to the right, to the left and downward extends the left ventricular cavity; note its thick walls, including the interventricular septum. The left atrial cavity extends to the left and upward. Same view, *Hardened specimen, opened (I)*: 46, arch of aorta, 47, lt. auricular appendage, 48, ascending aorta; 49, calcareous deposits around aortic orifice, 50, pulm. artery and portion of conus, partly dissected off and pulled away, 51, lt. ventricular cavity, outflow tract, 52, lt. ventricular cavity, inflow tract, 53, mitral orifice, 54, descending aorta, 55, lt. atrium.

*Comment*: The clinical picture was characteristic of aortic stenosis. The ECG showed evidence of left ventricular strain. There were noted radiologically lt. ventricular enlargement, calcareous aortic valve disease, and tortuosity of the descending thoracic aorta. Compensation was remarkable for a number of years. While the coronary flow must have been inadequate, angina pectoris was not noted at any time, and cardiac asthma came only as a terminal event.



FIG 109—Graphic registration of movements of silhouette borders in a case of aortic regurgitation. Simultaneous registration by means of multiple interrupted slit kymography. The black horizontal bars indicate level of different slits. Graphs appear in corresponding vertical bars. Respective contours from rt to lt: rt cardiac, rt vascular, pulmonary arch, aortic knob, lt upper ventricular, lt lower ventricular. An increased amplitude of



FIGS 200, 201, 202.—57 yrs, M Well up to age 43 when he served as soldier. Infectious state during which he continued military exercise. This was followed by progressive dyspnea, heart palpitation and pulsations of veins of neck. Cyanosis, marked positive venous and liver pulse. No cardiac murmur. Systolic retraction over several ribs and intercostal spaces at lt. B P 115/80. E.C.G.: auricular fibrillation; rt. axis dev. Diagnosis: postendocarditic tricuspid regurgitation. *Ant. view*: large silhouette. Prominence of contour superior vena cava (upper rt. wh. arrow). Spherical rt. cardiac



contour, large rt atrium. Absence of congestive lung failure. Fluoroscopy: Pulsations along rt and greater part of lt cardiac contours vigorous (7 mm), that of aortic knob and lt lower pole region small. This indicates that greater part of lt cardiac contour is formed by rt ventricle. Contour of superior vena cava prominent (wh arrow). *Rt. ant. obl view*: Ventrally, mass of heart encroaches upon retrosternal area; marked enlargement of rt ventricle (wh arrow), pulsations increased here. Dorsally, caudal half of retrocardiac space obliterated, enlargement of rt atrium (bl. arrow). *Lt ant obl view*: aneurysmal dilatation of superior vena cava (wh arrow), aortic arch just visible (bl. arrow). Pulsations large ventrally (rt ventricle), small dorsally (lt ventricle). Postmortem: marked hypertrophy and dilatation of rt ventricle. Lt ventricle not visible in situ. Rt. atrium size of fist. Tricuspid orifice much dilated without endocarditic changes. Superior vena cava at entrance into rt. atrium 8.5 cm in size. No valvular lesion, no pulmonary artery sclerosis; no microscopic

duration. . . . . a relatively long

of a second the lateral movement of the aortic knob and follows by a fraction of a second the medial movement of the left lower cardiac contour. Thus, when the left lower cardiac contour has already started its lateral movement, the right cardiac contour reaches its peak on the tracing. It seems possible that the lateral movement of the right cardiac contour is caused by the sudden distention of the bulb of the aorta.

Exaggerated pulsations have not been noted in instances of so-called functional aortic regurgitation as occasionally observed in the course of severe

lated for such cases.

Simultaneous with the marked systolic diminution of the silhouette, a marked depression of the anterior chest wall may be observed. Since an abnormally large quantity of blood flows, during systole, from the dilated



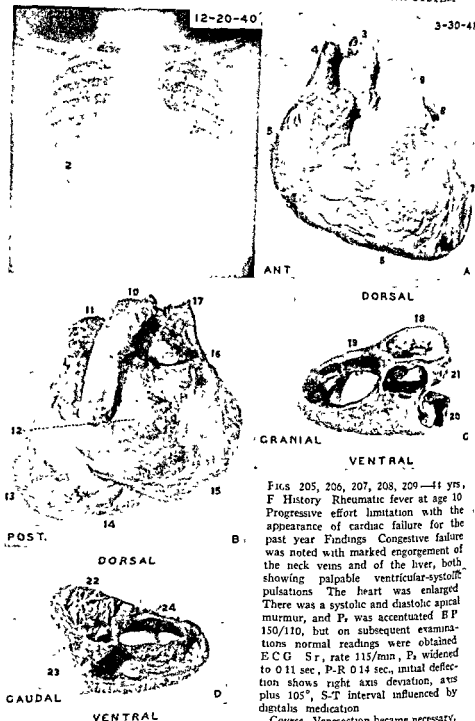
FIGS. 203, 204—18 yrs, M Tonsillitis and rheumatic fever at age 14 Congestive failure since age 17. Slight cyanosis and subicterus. Precordial bulge Systolic thrill and murmur at apex, transmitted to axilla  $P_2$  accentuated, systolic murmur at lower sternal area B.P. 102/80 Liver enlarged with systolic regurgitation murmur and systolic pulse ECG n.s.r., rt axis dev *Ant view.* large silhouette, mitral configuration Prominent arch at middle height and lt (wh arrow) conus arteriosus Prominence of rt atrial contour at rt (lower bl arrow) Hilar vessels insignificantly enlarged (upper bl arrow), absence of definite congestive lung failure *Rt ant obl view* mass of heart more ventral Caudal portion of posterior mediastinum overlapped at level of rt atrium (lower bl. arrow) Vascular shadows project over middle height of posterior mediastinum (upper bl arrow) Postmortem. mitral stenosis, mitral regurgitation, slight Tricuspid regurgitation Recurrent endocarditis of mitral, tricuspid and aortic valves Marked degree of rt ventricular and atrial hypertrophy and dilatation Enlargement of lt atrium Recent pericarditis with beginning obliteration of pericardial cavity

left ventricle into the arteries and since the bulk of it leaves the thoracic cavity, a rapid fall in the intrathoracic pressure results; and since the increased aspiration of air and influx of venous blood is not sufficient to compensate for this fall in pressure, it is compensated for by a reduction in the volume of the chest cavity resulting in a flattening of its curvature

In the presence of isolated stenosis of the aortic orifice one notes a relatively slow systolic change, tardus in type, in the shape of the left lower cardiac contour and the aortic shadow

Pulmonic. Stenosis is discussed in chapter XII, regurgitation in chapter X.

Tricuspid. Lesions affecting this orifice exclusively are very rare. Their combination with the common types of rheumatic valvular disease, however, occurs in about 20% of such cases and is discussed below. Both stenosis and regurgitation lead to an enormous enlargement of the right atrium; the right



FIGS 205, 206, 207, 208, 209—41 yrs, F History Rheumatic fever at age 10 Progressive effort limitation with the appearance of cardiac failure for the past year Findings Congestive failure was noted with marked engorgement of the neck veins and of the liver, both showing palpable ventricular-systolic pulsations The heart was enlarged There was a systolic and diastolic apical murmur, and P<sub>2</sub> was accentuated BP 150/110, but on subsequent examinations normal readings were obtained ECG S<sub>r</sub>, rate 115/min, P<sub>2</sub> widened to 0.11 sec, P-R 0.14 sec, initial deflection shows right axis deviation, axis plus 105°, S-T interval influenced by digitalis medication

*Course* Venesection became necessary, mercurial compounds were given intravenously, and digitalis orally Findings BP 130/90 The circulation time for arm to tongue (saccharin) was 40 sec, and for arm to lung (ether) 22 sec The laboratory findings for the blood were essentially normal Soon afterwards auricular fibrillation developed and became permanent.

ventricle remains relatively small in the presence of stenosis and is very large in the presence of regurgitation.

The right cardiac contour bulges almost spherically when a well marked tricuspid lesion is present, and the shadow of the superior vena cava is definitely widened and may even reveal a convex bulge. An abnormal widening of the vascular shadow caudal of the medial left clavicular portion may be present and is produced by the left innominate vein. The caudal portion of the retrocardiac space, as visualized in the right anterior oblique view, is encroached upon; this corresponds to the level of the right atrium. The left atrium is rarely very large but more commonly found to be smaller than one would expect in the presence of an associated mitral valvular lesion. The diminished load on the lesser circulation accounts for this finding. The same factor is responsible for the relatively small degree of congestion in the lung fields in these cases. When an organic, compensated tricuspid regurgitation is present, one notes two interesting facts in the anterior view. First, the basal (cranial) and middle portions of the left cardiac contour reveal an increased amplitude of pulsations which is also noted along the shadow of the superior vena cava but neither at the aortic knob nor at the left lower pole region. This proves that the right ventricle forms a considerable part of the left cardiac contour. Second, a slight, pulsatory movement may be noted along the contour of the right leaf of the diaphragm, indicating the systolic expansion of the liver, such a liver pulse is, however, not characteristic of tricuspid regurgitation alone since it is also found with several other cardiac lesions.

Experimentally produced tricuspid regurgitation shows an immediate widen-

*Ant view.* Moderate degree of lt-convex scoliosis is noted. The silhouette is enlarged. Note 1, prominence of pulmonic arch, 2, increased convexity of the rt atrial border. The lung fields reveal engorgement. Rt ant obl view. The lt atrium is considerably enlarged.

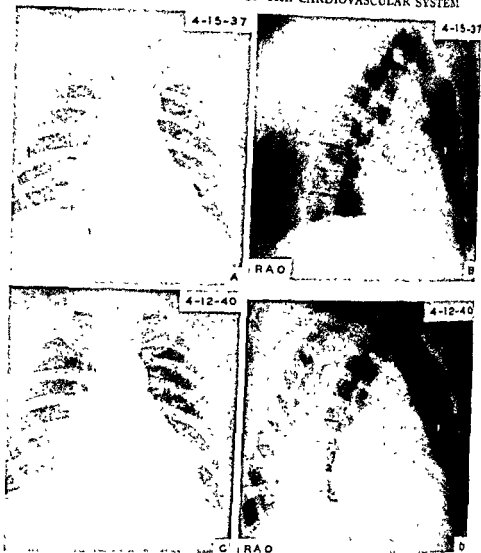
*Course:* The degree of congestive failure was temporarily unproved. The expansile liver pulsations continued. The patient developed a right-sided flaccid hemiplegia, fever, swallowing difficulties. Cheyne-Stokes respiration appeared. Death occurred 4 days later.

*Postmortem.* There was an embolus in the lt mid-cerebral artery. The weight of the heart was 500 gms. It was dilated, particularly in the rt atrial portion, and was hypertrophied. The mitral ostium was stenosed, and the leaflets heavily calcified. One tricuspid leaflet was definitely fibrosed and rolled in. The pulmonary artery was enlarged. Microscopic: Mitral and tricuspid leaflets showed fibrosis and small round cell infiltration; the former was also calcified.

*Hardened specimen.* Ant view (A) 3, arch of aorta, 4, sup vena cava, 5, rt. atrium, 6, rt ventricle; 7, lt ventricle, 8, lt auricular appendage, 9, pulm artery. Post. view (B) 10, arch of aorta; 11, lt main branch of pulm. art, 12, lt atrium, 13, lt. ventricle; 14, rt ventricle, 15, rt atrium, 16, lt main branch of pulm art, 17, sup vena cava. The great vessels and the walls of the four cavities were removed. The remaining base of the heart, with the valve orifices, was placed in an oblique and slanting position approximating the intravital position. Cranial view (C) 18, stenosed and calcified mitral orifice; 19, tricuspid orifice, 20, pulmonic orifice, 21, aortic orifice. Caudal view (D) 22, stenosed mitral

atrium, as visualized in the roentgenogram, is evident in the anatomic specimen. The appearance and topographic relationship of the valve orifices is demonstrated by proper anatomical dissection.





Figs. 210, 211, 212, 213—47, 50 and 52½ yrs, M History: Negative for rheumatism Patient was known to have hypertension and a slight degree of albuminuria at times, the fluid intake was excessive Sleep good, no headache, hardly any effort limitation on activity Findings (age 47) Wt. 82 7 kg. (182 lb) No evidence for failure The apical thrust was slightly displaced down and out, not heaving A short presystolic and diastolic apical murmur was heard,  $P_2$  was not accentuated, no basal murmur. Rhythm regular, rate 66/min. B.P. 185/95. Fundi Retinal arteriolar sclerosis, grade I, hypertensive type, with moderate attenuation of all, and spasticity of both inferior nasal, arterioles; very little

ECG. N.s.r.,  
its depressed  
nn negative,  
blood cells,

good concentration.

*Ant. view (A):* Normal appearance of the lung fields The heart is slightly enlarged in the lt. ventricular portion *Rt. ant obl view (B):* 1, slight prominence of conus of rt. ventricle; 2, practically no enlargement of lt atrium dorsally.

*Course:* A fairly good prognosis was given There were advised fluid limitation, a gradual diminution of physical activities in the years ahead, and medication with xanthin

ing of the upper right portion of the silhouette, which corresponds to the area of the superior vena cava.

**Combined Lesions.** The predominance of the respective roentgenologic characteristics depends upon several factors: the severity of the lesion, the time relationship of the onset and development of these lesions, the constitutional type of the patient and the possibility of other, associated cardiovascular disease. The presence of an aortic regurgitation modifies the roentgen appearance of a mitral heart in the following manner. The aortic knob is

products at times. Two yrs later the patient noted he had lost his "pep," and, although active and playing golf, he had to "take things easier," but there was no definite effort dyspnea. The family physician had noted a complete irregularity of the heart, and had prescribed digitalis. The fluid intake was still excessive,  $4\frac{1}{2}$  liters (5 qts.) in 24 hrs. Findings (age 50): No evidence for failure. The heart was enlarged, and auscultatory findings were the same as previously. Rhythm irregular, vr 86, pr 80/min B.P. 205/105. Fundi: Retinal arteriolar sclerosis grade II, post-spastic type, numerous small localized and several large areas of arteriolar narrowing, very little increased visibility of the reflex stripe, beginning arterio-venous compression, no edema, no retinitis, definite progression of the vascular lesion with evidence of previous vaso-spastic episodes, which are now less active than before. ECG: Atrial fibrillation, lt axis dev. less marked than previously, T deflections influenced by digitalis medication. Blood: Urea n. 14 mgm %, cholesterol 380 mgm %. Urine: Trace of albumin, no casts, a few red blood cells, good concentration. Fluoroscopy: Normal appearance of lung fields. As compared to findings 3 yrs previous: a) Minimal increase in heart size, 0.2 cm on both the oblique and broad diameters, b) slight increase in the size of the lt atrium. Recommendations were: Some curtailment of physical activities, limitation of fluids and salt, a trial with a diet low in cholesterol, digitalis, and a vacation in Florida. 2 mos later, while vacationing in Florida, he overate, went to the races, stayed up late, etc. The systolic B.P. rose to 240, and a slight attack of cardiac asthma occurred. After a few days of bed rest and medication with dilauid, he exerted himself again, and an attack of pulmonary edema occurred from which he recovered quickly. He was placed on a strict regimen for one month, then re-examined. Findings (age 50 $\frac{1}{4}$ ): Wt 70 kg (154 lb). No evidence for failure. The heart had further enlarged and, in addition to the apical diastolic murmur, there was now heard a hollow diastolic murmur to the left of the sternum, and P<sub>2</sub> was moderately accentuated. VR 70, PR 68/min. Radial pulse slightly collapsing. B.P. 185/80. ECG: Unchanged. Blood: Urea n 19 mgm %, cholesterol 283 mgm %. Urine Spec gr 1023, 12 hr Addis count reveals vol 980 cm<sup>3</sup>, rbc 10 mill, wbc 12 mill, casts  $\frac{3}{4}$  mill, protein 90 mgm %.

**Ant view (C):** A slight increase of the vascular pattern in the lung fields is noted. The heart is definitely enlarged in both lt and rt ventricular portions. **Rt ant obl. view (D):** 1, definite prominence of conus of rt. ventricle; 2, enlargement of lt. atrium dorsally. **Fluoroscopy.** As compared to the findings of 3 mos ago: a) Definite increase in heart size; the oblique diameter increased by 2.3 cm, the broad diameter by 1.6 cm. b) Further increase in the size of the lt atrium.

**Course:** In addition to medication and limitation of fluid and salt, the patient was placed on strict curtailment of physical activity. On this program he has been doing satisfactorily for an observation period of 2 $\frac{1}{2}$  yrs. Examination at that time (age 52 $\frac{1}{2}$  yrs.) showed the clinical and laboratory findings unchanged. The size of the heart, as measured orthodiagraphically, showed an increase of not more than 0.4 cm in the oblique diameter.

**Comment:** This patient suffered from hypertensive and valvular disease, the latter probably of the atherosclerotic variety. For a period of 3 yrs he did quite well, although some degree of effort limitation made its appearance. Left ventricular failure came on quite suddenly. This was probably due to improper habits plus the fact that an aortic regurgitation had developed in addition to the mitral stenosis present. A definite progression in the heart size was observed. The urinary findings, in the absence of congestive failure, indicated that the patient had entered the "nephritic" phase of nephrosclerosis.

better visualized and reveals some increase in the amplitude of pulsations. In those instances in which the presence of a rather steep course of the left lower contour indicates the prevalence of mitral stenosis, the pulsations, instead of being almost absent, are here easily seen. The presence of a mitral valvular lesion modifies the roentgen appearance of an aortic heart in the following manner. The waist of the heart is not so deep, the left atrium reveals enlargement in the oblique views and the vascular structures in the lung fields are more prominent.

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## CHAPTER IX

### DISEASES OF THE AORTA

Changes in the volume, density and pulsations of the aorta represent the chief roentgenological manifestations of any of the diseases affecting it.

The diagnosis of widening of the aortic shadow should not be made until there has been a careful analysis of the various views and an evaluation of clinical findings, viz, age, body weight, blood pressure, and presence or absence of aortic valvular lesion.

A formal description will be given first, to be followed by a discussion of the different etiological factors.

**Roentgenologic Findings. WIDENING AND ELONGATION.** Under normal conditions, in the anterior view, a vertical tangential line, which is drawn through the right vascular contour, is found to be medial to a similar line drawn through the right cardiac contour. Occasionally this distance may be zero and the aorta still be within normal limits but this occurs only in older people and in the presence of high blood pressure. Whenever the tangential line through the vascular contour is found to be lateral to the line corresponding to the right cardiac contour, fusiform dilatation is present. However, this dilatation may be only dynamic in character. With an adequate roentgen shadow can be demonstrated, the aorta bulges in addition its density is

greater than that of the atrium. The notch which separates the right vascular and cardiac contours is displaced caudad when the aorta is enlarged and as a result the right cardiac border is shorter than normal. The left anterior oblique view best demonstrates the presence of a moderate bulging where normally a mildly curved contour is noted. Under normal circumstances the cephalic portion of the vascular contour at the right is never convexly outlined. If this condition is found, the diagnosis of enlargement of the aorta at the first portion of the arch is definitely justified. Cephalic extension of the arch to the level of the jugular notch usually indicates dilatation but the vessel may be seen at this level when kyphosis of the lower cervical and upper dorsal spine exist. This is especially so in the aged. Broadening of the distal portion of the arch and of the very beginning of the descending aorta is expressed by an increase in the figures for the chord and radius of the aortic semicircle. An increase in the left horizontal dimension of the aortic shadow is noted when the descending aorta is widened but, since this measurement is increased if the aorta lies in the paravertebral position, it is necessary to take additional views to differentiate elongation and dilatation.

A widening of the aortic shadow is commonly associated with an expansion of the vascular shadow above it. The wider arch is responsible for this appearance though occasionally one or more of the brachio-cephalic vessels may be considerably enlarged. During fluoroscopy of stout individuals whose ventral chest wall slopes considerably, one should not be misled by a shadow

in the superior mid-portion of the chest which simulates intra-thoracic goiter. This shadow in reality is produced by extrathoracic fatty tissue located above the level of the clavicles in the neck or by an enlarged thyroid gland. The shadow will disappear during fluoroscopy if the patient leans forward and it is therefore not visualized on the film since the ventral chest projection is taken in this position.

Elongation of the aorta is characterized in the anterior view by three findings. The first is an abnormally long distance from the level of the right cardiovascular junction to the level at which the cephalic aspect of the aortic knob deviates medially. The second finding is an abnormal prominence of the aortic knob cephalically and to the left, either projecting toward the caudal aspect of the left clavicle or toward the cephalic aspect of the sternoclavicular junction. The size of the knob itself is unchanged unless there is an associated dilatation present. Sometimes the commencement of the descending aorta forms the left border of the aortic shadow and the more mesially situated left border of the arch is visualized inside. The third finding is an abnormal prominence of the descending aorta into the left or, very rarely, into the right lung field. The lateral contour of the aorta is curved and intersects the cardiac contour at different levels for individual cases, most commonly at the auriculo-ventricular junction. Below the level of intersection the vessel is situated more mesially, dorsad to the cardiac silhouette. The medial contour of the descending aorta becomes visible within the lung field only with extreme degrees of elongation. As compared with the density cast by the ascending aorta, the shadow of the descending aorta is definitely less. In the main, the difference in the object-film (or screen) distance accounts for this. Simple elongation is differentiated from a fusiform aneurysm by the use of additional views in the oblique positions.

The left anterior oblique view reveals two characteristic findings. The arch displays a wider swing, the type of curve resembling a circle, there is considerable overlapping with the vertebral shadow, and the window of the aorta is large. The middle portion of the descending aorta swings far dorsad, its caudal portion ventrad, thus a marked curve or even a kink at the inner contour results. Just below the level of the arch, an indentation of the lateral wall of the descending aorta is sometimes seen. This indentation might be mistaken for narrowing of the aorta as is present in coarctation but the latter condition can be ruled out by lack of rib erosion and clinical study.

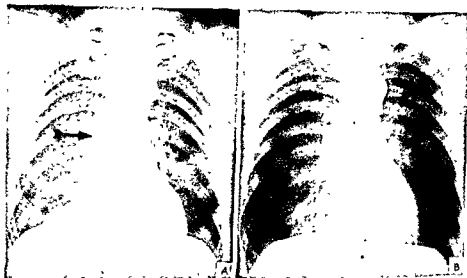
becomes visible

The best explanation for the abnormal course of the aorta is the conception that the vessel has become too long between its two points of fixation, the heart and the diaphragm.

In the presence of elongation and dilatation of the aorta, particularly of the atheromatous type as encountered in older people, the course of the esophagus is likely to show the following features: 1) the aortic bed at the level of the arch is deep and the left circumference of the esophageal wall extends in a diverticular fashion around a part of the caudal portion of the arch, this ap-

pearance has been likened to a ball in a socket; 2) it follows rather closely the deviation of the vessel and is therefore often found to the left of the midline; 3) it bends around the aorta at the midthoracic level. Subsequent examination in the lateral view reveals a marked convexity ventrally and this has been erroneously interpreted as evidence of the presence of an aneurysm of the descending aorta.

**INCREASED DENSITY.** The finding of an increased density of the aortic



FIGS. 214, 215 — 51 yrs., M. History Penile lesion 10 yrs ago that was treated. Complaints referable to gastric ulcer. Findings Mild emphysema was noted, there were no murmurs and A<sub>2</sub> was slightly accentuated. The B P varied at different times between 115 and 150 systolic and 60 to 100 diastolic. Wassermann negative. ECG - S r, moderate degree of lt ax dev.

**Roentgenograms** A (taken during tidal respiration). A moderate prominence of the ascending aorta is noted (arrow). B (taken at the end of deep inspiration). The shape of the silhouette has changed and, in particular, the ascending aorta is not prominent.

**Comment:** Presumably an instance of an asymptomatic syphilitic aortitis. Note the disadvantage for the roentgenologic diagnosis of aortic disease with the roentgenogram taken at the end of deep inspiration.

shadow is based upon a subjective impression which necessarily will have to be replaced by the objective method of densitometric determination. The evaluation

of the degree of contrast. This contrast is in the presence of the pres-

is to compare, by fluoroscopy, both the density of the vascular (aortic) and ventricular area. Provided that cardiac enlargement is absent, the aortic area is less dense under normal conditions. The aortic shadow is definitely increased in opacity when, in the left anterior oblique view, the contours of the arch are easily noted within the transparency of the tracheal lumen and when either the dorsal or both contours of the descending aorta are visible throughout its entire length. Intrinsic factors which influence the degree of density are the diam-

eter of the blood column and the condition of the wall (thickness and physico-chemical character of the constituent parts). The effect of changes in the wall on the degree of density may be easily proved. First, a normal-sized aortic shadow may be very dense. Second, a widened aortic shadow for instance, in the course of one year, may become denser without any further progress in the size of the vessel. Not until lime salt deposits are present to a considerable degree and especially when they are arranged in plaques will they be detected roentgenologically and usually only when there is a favorable summation of the shadows in the direction of projection (tangential effect). The region of the aortic knob may thus serve as an example. It is exceptional to note an inhomogeneous mottled appearance of the aortic shadow, and extensive degrees of an encasing type of calcification are likewise rarely observed. Lime-salt deposits appear sharply defined and are noted a few mm within the vascular contour. The presence of a sufficient amount and extension in the wall of the ascending aorta permits correct measurement of the lumen of this vessel and visualization of the inner and outer contours. With a fluoroscopic study of pulsation there is revealed, at least for this type of vessel, a considerable shift as a whole, best observed during the systolic phase



FIG 216—47 yrs, M Known to have valvular lesion for 14 yrs Pulsations to rt of sternum, site of systolic and diastolic thrill and maximum systolic and diastolic murmur BP 205/80 ECG: left axis dev, T<sub>1,2</sub> negative Wassermann positive *Ant view* typical aortic configuration, L 17.7, B 12.6, T 17.3, Th 26.2 cm Marked widening of aortic shadow, particularly along rt cranial contour Fluoroscopy shows amplitude of pulsations increased along all contours Course: sudden death. Postmortem left ventricular wall thickness 3 cm; syphilitic aortitis affecting greater part of thoracic aorta with saccular dilatation of ascending portion and marked rigidity of wall, stenosis of coronary artery orifices and syphilitic aortic regurgitation. Roentgen examination added nothing to clinical diagnosis Marked pulsations along aortic contours are not necessarily explained by wall distension only but may be caused by a shift of the vessel as a whole

**Etiological Consideration SYPHILIS** The diagnosis of the early stage seems to belong entirely to the clinician. The following, general statement, with the understanding that certain exceptions exist, seems justified: the more experienced the clinician, the less the roentgenogram is needed for diagnosis at that stage of the disease. There is no close correlation between the clinical signs and symptoms and the degree of dilatation of the vessel as revealed by the roentgen rays and by the anatomical study. The characteristic finding of a highpitched, musical-metallic second aortic sound, preceded by a short systolic murmur is often associated with entirely normal roentgenological findings

The mere fact that a postmortem study may reveal the syphilitic aorta to be



FIGS 217, 218, 219—55 yrs, M. Syphilitic infection at age 25. Fair compensation, retrosternal oppression. Aortitis with aortic regurgitation. B P. 160/45. Wassermann positive. *Ant. view*: enlargement of heart, predominantly to lt. Aortic shadow diffusely widened and plump in appearance. Lime salt deposits noted along contour of ascending aorta (wh arrow). *Rt. ant. obl. view*: lime salt deposits outline fusiform dilatation of ascending aorta (wh arrows); ventral contour of lt. bronchus (bl arrow). It is obvious that an attempted mea-



RAO



LAO

urement between ventral contours of ascending aorta and bronchus would give entirely erroneous values as to size of aorta. Note how far caudally root of aorta descends into cardiac shadow. Ventral contour desc. aorta (short bl arrow). *Lt. ant. obl. view*: cardiac silhouette extends far into shadow of spine (lt. ventricular enlargement). Contours of widened ascending aorta clearly visualized (wh arrows). Ventral and dorsal contours of trachea (bl short arrows). From Roesler, H, in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System, ed. by Ross Golden, 1936, p. 239-241. Courtesy Th. Nelson & Sons, New York.

of normal diameter and normal thickness, indicates that it may well be impossible to establish during life a positive diagnosis by roentgen rays. Likewise it may be a difficult matter at necropsy to distinguish between atheroma of the aorta and syphilitic aortitis because syphilitic aortitis is very frequently accompanied by atheromatous changes in the aorta, and this is not limited to patients of advanced years.

A diffusely dilated aorta, occurring under the age of 45, in the absence of aortic regurgitation and of hypertension present or past, and with normal

tion and yet disease in this region may result in a malignant course by producing stenosis of the coronary orifices. The visible part of the ascending portion is said to reveal increased pulsations during the stage which precedes cicatrization. This sign needs further study. Aortic valvular regurgitation must be carefully excluded and the examination repeated in order to overcome the patient's emotional reaction.

A markedly convex prominence of the ascending aorta, together with increased pulsations limited to this area, are quite characteristic findings. The vascular contour is definitely convexly curved. The use of the left anterior oblique view is quite indispensable. A fusiform dilatation in the right anterior oblique view with cephalically convergent borders can be diagnosed only when the density of the aortic shadow is simultaneously increased, and this is affirmed when the borders are outlined by lime salt deposits. Its size is thus determined by a comparison with the

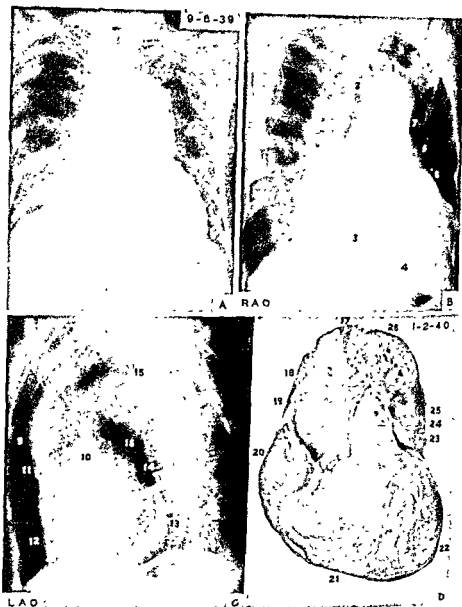


FIG. 220—About 50 yrs, M. Retrosternal burning sensation. Upper sternal area flat on percussion. A, high pitched Wassermann positive. Syphilitic aortitis. Ant view cardiac silhouette not enlarged, aortic configuration, slight. Diffuse widening of aortic shadow, described as chimney type. Cavitation in rt. lung. Course: death from pulmonary tuberculosis.

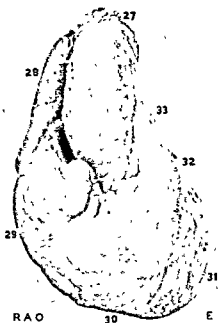
size of the arch (barium method) and will demonstrate a greater difference than encountered normally (see chapter IV). In other instances a moderate degree of diffuse widening of the entire aortic shadow is noted which, in the anterior view, produces the impression of a chimney situated on the base of the heart.

Syphilitic aortitis has been too often diagnosed in children since in them it is a rare anatomical finding.

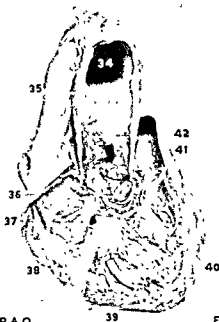
Dilatation of the aorta progresses slowly. Search for the appearance of aortic valvular regurgitation or for a marked increase in the blood pressure is indicated whenever progression is observed to take place within a relatively short time. Marked elongation (tortuosity) is not a characteristic finding of syphilitic aortitis and must be ascribed to associated changes in connection with greater age, atherosclerosis and hypertension. Widening of the vascular lumen has already been stated to cause a moderate increase in density. A marked increase in density must be ascribed either to a pathological thickening of the wall, occasionally combined with media calcification, or to calcifying



FIGS 221, 222, 223, 224, 225, 226, 227—64 yrs, M History. Progressive exertional and nocturnal dyspnea during the past 5 yrs Slight ankle edema at times Findings Wt 51 kg (112 lb) Tabes dorsalis was present Cheyne-Stokes respiration, slight enlargement of the liver, emphysema, and a few basal rales were noted The heart was enlarged and hypertrophied, the apical thrust was in the 5th isp in ant axillary line, the upper sternal area was dull to percussion, the apical sounds dull, a short systolic murmur was noted at base and apex, and A<sub>2</sub> had a high pitch B.P. 130/65 Fundi: Atherosclerosis of choroidal vessels, senile fibrosis of retinal arterioles Blood and spinal Wassermann positive Blood count and chemistry normal ECG Left bundle branch block with at times a) sinus rhythm with premature beats of auricular, nodal, and ventricular origin, b) auricular fibrillation



RAO



E RAO

F



LAO

G

*Ant view (A)* Heart enlarged, aorta diffusely dilated *Rt ant obl view (B)* 1, arch of aorta, dilated, 2, sup vena cava, 3, rt atrium, 4, rt ventricle, 5, lt ventricle, 6, 6, outflow tract of rt ventricle and pulm artery, prominent, 7, ascending aorta, dilated, 8, trachea *Lt ant obl view (C)* 9, ascending aorta, dilated, 10, bifurcation of pulm artery, 11, rt auricular appendage, 12, rt ventricle, 13, lt ventricle, enlarged, 14, lt atrium, 15, arch of aorta, dilated, 16, lt main bronchus

*Course* Failure progressed 12 days before death a basal diastolic murmur was perceived for the first time, short and harsh in character, it was also heard over the subclavian artery on the right B.P. 124/60. Pulmonary infarction developed Death 4 mos following the radiologic study.

*Postmortem:* The weight of the heart together with the aorta was 750 gms Both ventricles were enlarged The pericardium was adherent

at the base of the aorta The aorta was dilated throughout, its diameter at the arch measured 6 cm., a typical syphilitic aortitis with associated mild atherosclerosis was noted, the aortic cusps were slightly thickened, and the ostia of the coronary arteries were not narrowed, (the arteries themselves were not dissected) The organs were congested, and an



atheroma which is likely to be present in the older age group, or to a combination of both factors. The contours of the aortic shadow are sharply defined provided the exposure time is sufficiently short. An irregularity in outline of the aortic shadow is often due to pleuro-mediastinal disease. Changes caused by periaortitis seem to be too significant to be considered.

The aortic arch impression on the esophagus is hardly changed if the disease

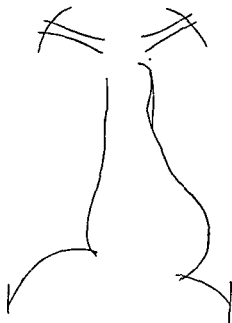


FIG 228—43 yrs, M Syphilitic infection at age 24. Apical thrust heaving A, greatly accentuated B P. 240/140 Spinal fluid Wassermann positive *Ant. view*: vertical position of silhouette with aortic configuration L 13.3, B 10.3, T 11.8, Th 21.7 cm Lt lower contour markedly rounded. Aortic shadow small. *Obl views* (illustrations omitted): normal appearance of aortic shadow. Death caused by apoplexy. Postmortem: syphilitic aortitis limited to supra-valvular part of aorta with stenosis of both coronary artery orifices. Marked hypertrophy of left ventricle. Atherosclerosis of abdominal aorta. Note normal aortic shadow despite hypertension and syphilitic aortitis. Unless calcification is present, supra-valvular portion of aorta is inaccessible in roentgen examination.

process affects the ascending aorta or the first portion of the arch. With the distal-dorsal portion of the arch involved the distance from the barium curve to the aortic knob surpasses normal figures.

**ATHEROSCLEROSIS** That part of the aorta which is commonly submitted to a roentgenological study, ascending, arch and upper descending portions, often

infarct at the base of the right lung was found. *Hardened specimen, ant view* (D) 17, vessels to neck, 18, sup vena cava, 19, ascending aorta, dilated, 20, rt atrium, 21, rt ventricle; 22, lt ventricle, 23, lt auricular appendage, 24, pulm artery, 25, descending thoracic aorta, dilated; 26, arch of aorta, dilated. *Rt ant obl view* (E) 27, arch of aorta, dilated, 28, sup vena cava, 29, rt atrium, 30, rt ventricle, 31, lt ventricle, 32, outflow tract of rt ventricle and pulm artery, prominent, 33, ascending aorta, dilated. Same specimen with *window section* (F). 34, arch of aorta, dilated, 35, sup vena cava, 36, aortic orifice, cusps nearly normal, 37, coronary sinus, 38, rt atrial cavity, 39, rt ventricular cavity, wall thickened, 40, lt ventricle, 41, pulm artery, 42, ascending aorta, dilated, wall thickened, intima puckered. *Lt ant obl view* (G) 43, ascending aorta, dilated, 44, bifurcation of pulm artery, 45, rt auricular appendage, 46, rt ventricle, 47, lt ventricle, enlarged, 48, lt atrium, 49, arch of aorta, dilated.

*Comment:* Cardiac enlargement and failure were probably on the basis of atherosclerosis rather than on the basis of the syphilitic aortitis, because the ostia of the coronary arteries were patent, and aortic regurgitation was not present until very late and then relative in nature. The diagnosis of syphilitic aortitis could be easily established by both clinical and radiologic examination.

reveals anatomically but little change as compared with the lower thoracic and abdominal portion.

Atherosclerosis, as a rule, leads neither to elongation nor to dilatation. The latter exists more frequently in the older age groups. The older the individual the more broadening may be expected. However, it must be remembered that the physiologically-large aorta in aged individuals is easily compatible with absence of atherosclerotic process. The induration of the wall alone, without

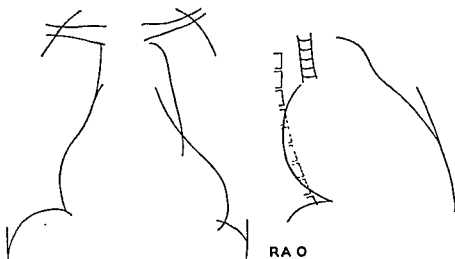
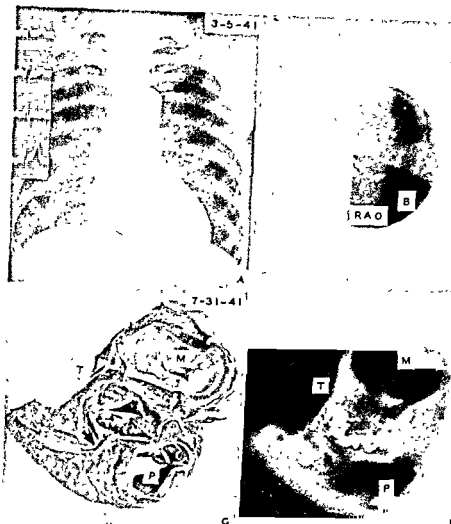


FIG 229—57 yrs, F Progressive cardiovascular failure since age 54 Mitral stenosis and aortic regurgitation BP 190/60 ECG auricular fibrillation Wassermann positive *Ant view.* marked enlargement of silhouette, combination of mitral and aortic configuration L 17.2, B 12.6, T 17.2, Th 25 cm Contour of descending aorta prominent, density of aortic shadow definitely increased Fluoroscopy showed increased pulsation of lt. lower contour *Rt ant obl view.* marked enlargement of lt atrium Postmortem. rheumatic mitral stenosis and aortic regurgitation, syphilitic aortitis of entire thoracic aorta; marked hypertrophy and dilatation of both ventricles and enlargement of lt atrium The presence of vascular syphilis had not been suspected, either clinically or roentgenologically

deposition of lime salts, does not influence the density of the aortic shadow, but an increase in the lumen of the vessel or in the thickness of the wall, or of both, and the presence of diffuse calcification do cause the aortic shadow to appear dense. Lime salt deposits are usually localized and can be well demonstrated, especially on tangential views. They have been noted in younger individuals and indicate a prematurity of this process. Combination with syphilitic aortitis occurs. It is well to point out that the association with, or absence of, syphilitic aortitis often can be decided only by a microscopic study.

The rigid aortic vessel is displaced during the act of swallowing and this may amount to more than 1 cm. at the level of the aortic arch. This test is performed with the patient's head elevated so that trachea and esophagus are stretched.

**RHEUMATISM** Aortitis and periaortitis have been described anatomically and a few roentgen ray findings are available, but conclusions must be deferred



FIGS 230, 231, 232, 233—48 and 50 yrs, M History Rheumatic fever at age 17. Onset of occasional fainting attacks a few yrs later Anginal syndrome and effort dyspnea for the past few mos Findings There was no evidence for cardiac hypertrophy and no thrill There was a rough systolic murmur at apex and base, the latter transmitted to the neck vessels, with a squeaky character over the lower sternum There was a faint diastolic midsternal murmur with  $A_2$  inaudible The rhythm was regular BP 92/70 Fluoroscopy revealed moderate prominence of the ascending aorta, heart at upper limit of normal size with rounding off of the lt ventricular contour and calcification of the aortic ring

Course: Observation over a period of 2 yrs showed little change except that a thrill appeared over the aortic area and that the lt ventricle increased in size. At no time was there congestive failure ECG. Sr, rate 72/min, P deflections notched, QRS slurred with lt. ax dev, T positive in I, II, CF.

Ant. view (A) An aortic configuration is noted, the lt. ventricular contour is rounded off and the ascending limb of the aorta is moderately prominent Rt. ant. obl. view (B—coned down, overpenetration); Calcification of the aortic ring area is noted Fluoroscopy showed dancing movements of these calcific deposits

Course: The patient died suddenly 5 mos later

Postmortem. The heart weighed 520 gms The lt ventricular wall measured up to 3.4

until cases are found in which these findings are corroborated. The possibility of a dynamic dilatation in connection with aortic valvular regurgitation and increased blood pressure should be borne in mind.

**CYSTIC MEDIAL NECROSIS.** Microscopic examination shows a widespread degeneration of both muscular and elastic tissues and the formation of small cystic areas. The etiology of the process is unknown. There may result a diffuse

**Aneurysm. THORACIC.** This is known to occur in about one-fifth of all cases affected by syphilitic aortitis and is observed in two forms, diffuse (cylindrical and fusiform) and as a saccular out-pouching. Fusiform aneurysms rarely reach the great size of the saccular variety. With the most common etiological factor, syphilis, the other adjacent parts of the aorta are usually also found to be diseased, resulting in some widening. The frequency of localization is greatest for the ascending portion, next follows the arch and finally the descending part. Combinations also occur; aneurysms affecting simultaneously the ascending and arch portion are rather common. The heart is either not influenced at all, is displaced, or changed in size and shape when any one of the following complications or combination of them is present, viz.: aortic valvular regurgitation, coronary artery disease, hypertension. There should be added the rare instances when an aneurysm compresses the pulmonary artery or ruptures either into it, the superior vena cava, or the right heart. The diagnosis is of importance, first of all from the viewpoint of prognosis. As to therapy, it may be stated that the antisiphilitic treatment does not bring about a change in the size of the aneurysm as has been often erroneously stated, but it may prevent further progression of the disease process. A fall in the

cm in thickness, and thin grayish strands ran through the myocardium, the thickness of the rt ventricular wall was up to 1.5 cm. The aortic ring was rigid and extremely hard. The aortic cusps were rigid, adherent, produced a marked degree of stenosis and some degree of regurgitation. Their aortic surface contained many hard nodules that extended from the free margin to the sinuses of Valsalva. The coronary orifices as well as the coronary arteries were widely patent with a few plaques in the latter. The root of the aorta measured 9 cm in width and there was a minimal number of yellowish plaques. The mitral leaflets and their chordae tendinae were slightly thickened. Microscopic: Heart muscle, patchy myosclerosis, ascending portion of aorta, presence of a marked amount of elastic tissue, occupying practically the entire medial coat. *Hardened specimen, base of heart (C)*. The tricuspid ring has been removed. The mitral, tricuspid and pulmonic orifices are indicated by the appropriate initials. The narrowed aortic orifice and the calcific deposits that cover a part of the cusps are visualized. The coronary arteries have been dissected out and are opened, lt (wh arrow) and rt (bl arrow). *Roentgenogram of same specimen (D)*. Note the extensive calcification of aortic ring and cusps and a few calcific plaques in the coronary arteries.

*Comment:* The aortic valvular lesion, mainly stenotic in character, was chiefly responsible for effort limitation. In fact, the only marked stenosis was at the aortic orifice.

blood pressure, however, may lead to some diminution in the size of the aneurysm. Also intra-aneurysmal thrombosis may cause a reduction in apparent size of the aneurysm.

Aneurysms of the thoracic aorta may be the cause of symptoms which are mistakenly referred to, for instance, bronchi, lungs or esophagus.

It should be borne in mind that not all aneurysms are demonstrable by roentgen ray methods. Small out-pouchings from the sinuses of Valsalva, which are directed towards either atrium or the right ventricle, and small pouches located in the concave or convex aspect of the arch, are examples. Generally speaking, the diagnosis is more difficult when the aneurysm is pedunculated,



FIGS 234, 235—80 yrs, M A, moderately accentuated B.P. normal. *Ant view:* Aortic configuration, marked elongation and tortuosity of aorta. Size adequate for this age (Upper bl arrow) medial contour of arch of aorta; (lower bl arrow) contour of pulmonary artery, (wh arrow) lateral contour of descending aorta. *Rt ant obl view:* ascending and descending aorta may be visualized separately because of increased tortuosity. Course of ascending aorta deviates ventrad. Ventral contour of distal portion of ascending aorta (wh. arrow), dorsal contour of thoracic descending aorta (upper bl arrow); kink along ventral contour of caudal portion of descending thoracic aorta (lower bl arrow)

or of very small or very large size, while relatively little difficulty is experienced with the medium sized aneurysms.

The fluoroscopic study of pulsations has its diagnostic limitations. Expansile pulsations may or may not be visible. Some of the factors which render these movements imperceptible are thickening and inelasticity of the vessel wall, presence of a thrombus in the sac, an unusually large aneurysm, a narrow communication between the vessel and the aneurysm, and a failing myocardium. If accessible to palpation, the clinical perception of these pulsations is much more impressive than the fluoroscopic aspect.

Lack of definition of the margins of the sac may be due to diminution in aeration of the surrounding lung because of extravasation of blood from the aneurysm or because of atelectasis. A large pleural effusion will interfere, also with visualization of the sac. The shadow of the sac is inseparable from that of the remainder of the aortic silhouette and, in certain positions, the continuity of the vessel and aneurysmal wall can be seen.

A careful analysis will demonstrate that, in the presence of large spindle and

saccular aneurysms, the widening of the aortic shadow is not limited to the sac, but extends over adjacent portions of the vessel or affects it in its whole course. In short, the underlying disease often affects the vessel over a greater distance than is apparent at first sight. Portions of the vessel are displaced beyond their normal positions because of the lengthening of the entire vessel (interpolation of the aneurysmal mass). Exceptions to this rule are found in



FIGS 236, 237, 238, 239—50 yrs, F Angina pectoris on effort A, moderately accentuated. B.P. 145/80 Chest Ant view lime salt deposits in aortic arch (bl arrow) Rt ant obl view extensive deposits in descending thoracic aorta, slight kinking along its course (bl arrow), definite prevertebral site Lt ant obl view extensive deposits in arch and descending thoracic aorta, slight kinking along its course (bl arrow)—Abdomen, lumbar spine region Lat view lime salt deposits noted (bl arrows), outlying course of abdominal aorta

aneurysms with the following etiologies: traumatic (external trauma or erosion by an adjacent tumor); mycotic-embolic, congenital (sinus of Valsalva).

The chief axis direction of aneurysms of the thoracic aorta is governed by two forces one is the direction of the systolic impact of the blood stream, many aneurysms developing as an elongation of the preceding aortic segment; the other is gravity Exceptions to this rule are represented by globu-

lar shaped aneurysms and those of enormous size. From the above statement it follows that the general direction of aneurysmal sacs depends on their location. If the proximal ascending portion of the aorta is involved, the sac is directed craniad, ventrad and to the right; if located at the distal ascending portion, it points craniad; if at the transverse limb, it lies in the horizontal plane; and if at the descending limb, it points caudad. These general principles are modified by the resistance of the bony chest wall structures and by the mass of the heart. The direction of the systolic impact of the blood stream is responsible for the preferential site along the outer contours of the curved portions of the aortic vessel.

Lime salt deposits are not uncommonly present; they are noted in the



FIGS. 240, 241—58 yrs, M Dizziness and headache since age 54. A, accentuated B.P. 190/110 Wassermann negative *Ant view*: silhouette moderately enlarged to Lt Contour of ascending aorta and aortic knob prominent. Marked elongation and tortuosity of descending aorta which deviates far into Lt lung field. Lateral contour (wh arrow) and medial contour are visualized. *Lt ant obl. view*: prominence of caudal portion of descending thoracic aorta dorsally (wh arrow). Barium-filled esophagus follows, in part, the course of the aorta.

form of oval or semicircular, sharply delineated, small and dense shell-like shadows lying a few millimeters inside the shadow contours. Individual variations in this distance depend upon the main site of calcification (intima-media) and upon the thickness of the adventitia, which may be relatively great because of associated periaortitis. These deposits are usually not sufficiently extensive to permit their visualization within more central portions of the shadow of the aneurysm. The demonstration along the contours is a summation effect produced in the tangential view. However, their presence conveys a marked density to the whole aneurysm.

An erosion of the spine should be looked for in the oblique and lateral views and often is easily accessible for the fluoroscopic diagnosis. The vertebral bodies are hollowed-out along the ventral and sometimes along the lateral aspects while the cranial and caudal cortical layers, together with the intervertebral discs, remain inviolate. Unless the aneurysm is quite large or the impact against the vertebrae is great, the spine exhibits a polycyclic contour. Ribs and sternum may likewise become eroded.

Since the root of the *ascending aorta* is not available for direct visualization, a small aneurysm in this region cannot be diagnosed roentgenologically. In



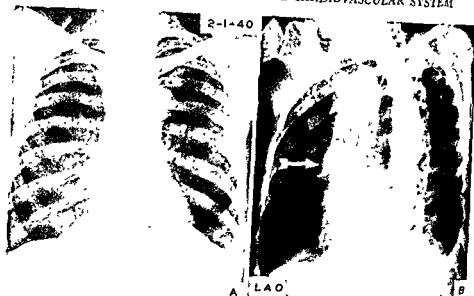
FIGS. 242, 243—40 yrs, M Compensation case following accident. No complaints referable to cardiovascular system. Abnormal pulsations 3rd rt intercostal space.  $A_2$  high pitched. B.P. 135/95. Wassermann positive. *Ant view* silhouette slightly enlarged to lt. Aortic knob moderately prominent. There is a round mass noted at intersection of rt cardiac and vascular contour (wh arrows). Fluoroscopy delineates expansile pulsations of this mass. *Rt. ant obl. view*: the mass projects into aortic shadow (wh arrows). Small aneurysm of the beginning portion of the ascending aorta.

exceptional cases, however, either a sufficient amount of calcification or some ventral out-pocketing will permit the direct demonstration of a denser area



FIGS. 244, 245—58 yrs, M Angina pectoris. Area of pulsation in second lt intercostal space. Aortic regurgitation with diastolic murmur best heard to lt of sternum. B.P. 160/55. Wassermann positive. *Ant view* calcified area noted caudad to aortic knob (wh arrow). It reveals expansive pulsations by fluoroscopy. Normal findings along pulmonary artery branches. *Rt. ant obl view* an aneurysmal, calcified pouch visualized ventrally (wh arrow). Calcification noted in course of aorta, ascending portion (ant and post bl arrow), arch (upper bl arrow). Syphilitic aortitis with aneurysm of ascending aorta, developing ventrad and to lt. This fact explains why diastolic murmur was so well heard to lt of midline. The presence of pulsations to lt of midline together with an abnormal pouch at same level may be found in pulmonary artery disease also. However, pulmonary artery regurgitation shows characteristic expansile pulsations of the hilus vessels which were not observed in this instance. From Roesler, H, in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System, ed by Ross Golden, 1936, p 243. Courtesy Th Nelson & Sons, New York.





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Figs 246, 247, 248—49 yrs, M. History: The patient was treated for syphilis on three occasions. Indigestion, anorexia and weight loss had developed during the past 9 mos., and radiologic studies revealed tumor of stomach. Wassermann positive. The operation revealed an infiltrating and unoperable tumor. Findings: Wt 59.1 kg (130 lb.). There was dullness to percussion over the upper sternal area. Rhythm regular, B.P. 115/70. Moderate degree of anemia.

is an additional rounded enlargement of the vascular tree in the region of the innominate artery. *Lt. ant. obl. view (B)*: A pouch-like prominence is noted, corresponding to the ascending portion of the aorta (wh arrow).

C

*Course.* Death six days later due to bronchopneumonia.

*Postmortem.* Reticulum cell sarcoma, invading the stomach. Obliteration of the pericardial cavity over the base of the aorta (luetic pericarditis). The heart was small, 12 x 10 x 6 cm, compressed from above downward, together with the aneurysm attached, it weighed 285 gms. The coronary artery ostia were open, the arteries themselves showed nonobstructive atherosclerosis. The aortic valves showed some adhesions at their point of attachment to the aorta and thickening of the leaflets at their attachment in the depth of the sinuses of Valsalva. A fusiform aneurysm started 1.5 cm above the line of attachment of the aortic valve cusps, involving the remainder of the ascending limb and almost the entire transverse limb of the arch of the aorta, measuring 8 x 10 cm. The wall was thin, with surface adhesions at the base of the aneurysm on the arch.

within the cardiovascular shadow. Aneurysms may arise from the sinuses of Valsalva. They are usually of syphilitic origin, but occasionally may be of mycotic or congenital origin. The left lateral sinus corresponds to the left (left posterior) aortic cusp and the orifice of the left coronary artery. When a bulge develops in its anterior half, it will be directed towards the pulmonic orifice and artery. The anterior sinus corresponds to the right (anterior) aortic cusp and the orifice of the right coronary artery. A bulge of its anterior half will develop against the interventricular septum and the conus of the right ventricle.

tures and will thus interfere with function. These aneurysms usually measure several cms. in diameter. Oblique and lateral views may establish the diagnosis, but observation limited to the anterior view may lead to failure in proper diagnosis. Such pouch-like prominences should be searched for along the cardio-aortic-pulmonic angle at about the level of the hiluses. With greater size, a prominence is noted in the anterior view at the level of the pulmonary conus and artery. In an exceptional case, a large sac develops which extends to the aortic knob, down to the diaphragm, overshadowing the whole left cardiac contour and displacing the heart to the right. This left sided location of an aortic aneurysm gives rise to clinical symptoms and signs to the left of the midline which simulate pulmonary artery disease and in case of associated aortic valvular regurgitation an erroneous diagnosis of pulmonary valvular regurgitation or rheumatic aortic valvular regurgitation may be made. Pressure on and rupture into the pulmonary artery lead to right ventricular enlargement. The typical large aneurysms of the ascending aorta extend into the right lung field, and partly or entirely overshadow the right cardiac contour, displace the heart caudad and to the left, and displace the aortic arch cranial and to the left. Right basal atelectasis and paresis of the right leaf of the diaphragm may be observed. Rarely does the aneurysm develop far enough dorsad to cause a displacement of the esophagus. The cardiovascular junction at the right is clearly visible when the distal portion of the ascending aorta is exclusively affected. The aortic knob is markedly denuded and displaced to the left. The main right bronchus is often noted to be elevated.

An aneurysmal dilatation of the *innominate artery* causes an asymmetrical widening of the superior mediastinal shadow to the right, the long axis of the aneurysm being parallel to the body axis. The lateral contour is oval or semicircular and its cephalic end fades out into the soft tissue shadows of the medial supraclavicular area. The relatively faint shadow of the superior vena cava, with its straight lateral contour, becomes clearly visible, this

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of the aorta and from the proximal 15 cm. of this artery. This aneurysm measured 6 cm. in diameter, the wall was thick and stiff, the lumen was almost completely filled by a laminated, organized thrombus. The remainder of the aorta showed plaques which were characteristic of syphilitic aortitis. *Hardened specimen (C)* Showing small heart, fusiform aneurysm of aorta and globular aneurysm of innominate artery.

*Comment.* An instance of syphilitic aortitis with aneurysms of the aorta and innominate artery. These findings were of no particular significance clinically.

vessel being displaced to the right. The shadow of the aneurysm fuses broadly with the shadow of the arch. Since the innominate artery lies in front of the trachea, the latter structure is commonly displaced dorsad and its ventral wall is compressed. Rarely is the trachea pushed to the left. The esophagus undergoes a displacement likewise. Differentiation from unilateral substernal thyroid is not difficult as the goiter displaces the arch of the aorta caudad and to the left, is wider in its cephalic portion, moves with the trachea and does not pulsate.

Aneurysms of the *aortic arch* cause the most pronounced subjective symptoms



FIG 249—63 yrs, M Syphilitic infection at age 21 Dullness over upper sternal area and rt sterno-clavicular region Thrill in rt supra- and infra-clavicular fossae Aortic regurgitation. BP rt 95/40, lt 125/45 ECG nsr, lt axis dev Ant. view. aortic configuration with enlargement of cardiac silhouette Vascular shadow widened, its rt contour formed by superior vena cava (middle rt wh. arrow), medial contour of ascending aorta visible (lower rt wh arrow) Extending from arch of aorta craniad and to rt, an oval mass is noted, equal in density to aorta, pulsating in fluoroscopy (upper rt wh arrow) It obviously displaces superior vena cava to rt Trachea not displaced Shadow of descending thoracic aorta prominent (lt wh arrow) Obl views (illustration omitted) abnormal mass is inseparable from aortic shadow. This indicates either an aneurysm of the innominate artery or of the aortic arch at origin of the innominate artery.

by interfering with the trachea, esophagus and laryngeal recurrent nerve. The ventral projection may show only a club-like widening of the cephalic mediastinal shadow or an abnormal density which corresponds in size to a small egg but the oblique and lateral views will probably demonstrate encroachment on the trachea and the esophagus. With a larger sized aneurysm, both trachea and esophagus are commonly displaced in an arc-like fashion, dorsad and to the right and are narrowed to different degrees. Occasionally the aneurysm does develop between the trachea and esophagus, separating these structures and displacing the former ventrad and the latter dorsad. Lateral views clearly reveal the encroachment on the cranial transparent retrosternal space. When the process affects the distal portion of the arch and the beginning of the descending aorta, the abnormal prominence replaces the aortic knob in the anterior view, and in the left anterior oblique view it projects into the shadow of the spine. The window of the aorta is narrowed in its dorsal aspect and the trachea and esophagus are displaced ventrad and their lumen is encroached on. If the aneurysm is large enough the lumen of the left main bronchus most information is obtained by the right posterior oblique view. Transverse portions are common and

Aneurysms of the *descending thoracic aorta* vary in appearance considerably, so that these variations must be analyzed separately. A fusiform enlargement may not be discerned in the ordinary anterior view but if the film is overexposed a nuclear dense shadow may be seen within the cardiac shadow. The right border of the aneurysm may overlap the right cardiac border so that the dense cardiac shadow is accompanied by a more transparent band-like shadow. Oblique and lateral views permit direct visualization of the bulging

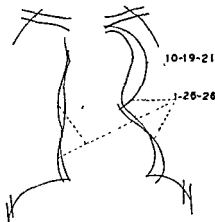


FIG 250—48 yrs, M. Dyspnea since age 36. Hoarseness, cough and difficulty in swallowing since age 45. Marked dullness over upper sternal area. Pulsations in jugum and in 3rd to 5th lt. intercostal spaces. Aortic regurgitation B P 135/60. Wassermann positive. Roentgen ray findings entirely unchanged for observation period of 4 years. Specific treatment not undergone. *Ant view*: cardiac silhouette moderately enlarged to lt and caudad. Three aneurysms: (a) arch (rt wh arrow), (b) cranial portion descending thoracic aorta (lt upper wh arrow), (c) ascending aorta with development ventrad and to lt (lt lower wh arrow). The difference in clarity of the lime salt deposits in (b) and (c) is explained by the different object-film distances. Course: death as result of rupture into esophagus. Post-mortem: syphilitic aortitis with three aneurysms, erosion of sternum.

FIG 251—48 yrs, M. Syphilitic infection at age 21. Slight degree of dyspnea on effort and occasional substernal pain since age 38. Relatively well. Two studies, 5 years apart. At second examination: no failure. Dullness in lt interscapular space and over upper sternal area. A<sub>2</sub> metallic in character. B P 180/90. Wassermann positive. *Ant view*: aortic shadows show diffuse enlargement and aneurysmal formation respectively. Aortic configuration

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ventral edge at the level of the middle third of the vessel or just above the diaphragm. Transmitted pulsations to and displacement of the barium-filled esophagus is also an aid in the diagnosis, particularly because the outline of these aneurysms is seldom wholly visible. A fusiform or saccular enlargement with marked enlargement of the bulging of the vessel

disappears. In other instances no shadow of the descending aorta is noted at

the left side but, as the oblique views reveal, it crosses the midline to the right and passes directly into an aneurysmal sac which is located in the dorsal portion of the right chest cavity. The esophagus is displaced ventrad and markedly to the left and, if the aneurysm is of sufficient size, the right cardiac border becomes invisible. Saccular aneurysms usually extend into the left lung field and are therefore easily visualized in the anterior view. When the sac is directed ventrad and located at the mid-portion of the descending lumb, bronchial stenosis must be looked for.

Finally, rupture of the aorta may lead to the formation of a sub-pleural, para-aortic hematoma. These sacs are not very sharply defined and are of less density than is to be expected in true aneurysms.

**ABDOMINAL.** There is little justification for separating aneurysms of the abdominal aorta from those affecting the lower portions of the descending thor-



FIGS 252, 253—62 yrs, M. Burning pain in back. Short systolic and occasional diastolic aortic murmur; A<sub>2</sub> of metallic character. Flatness in lt interscapular space. B.P. 190/00. Syphilitic aortitis, aneurysm, hypertensive disease. *Ant. view:* aortic configuration and lt-sided enlargement of silhouette. Diffuse widening of aortic shadow with bulging prominence in course of descending thoracic aorta (wh. arrow). Width of aortic arch 5 cm. (short bl. arrows). *Rt. post. obl. view:* aneurysmal pouch well visualized (rt wh. arrow). Lumbar salt deposits in ascending aorta (lt wh. arrow). Both contours of aortic arch noted (short bl. arrows). Smaller aneurysms in the upper and outer or posterior portion of the descending thoracic aorta are best studied in the rt. post. obl. view.

acic aorta. The diaphragm does not act as anatomic or functional barrier. As compared with aneurysms of the thoracic aorta, atherosclerosis is more often the etiological factor in abdominal aneurysms. The presence of such a mass in the abdomen necessitates the differential diagnosis from other abdominal tumors, especially those of the kidneys. Pain, chiefly of radicular character, is often present and may lead one's diagnostic thought towards cancer or ulcer of the stomach, gallbladder or renal colic, pancreatitis, tabetic crisis, or lumbago.

The fluoroscopic examination will lend enhanced diagnostic information by the use of two supplementary technics. First, by means of a barium meal: frequently this may reveal that the cardia end of the stomach is displaced ventrad, or ventrad and to the left, with the lesser curvature of the stomach indented in a shallow fashion. Second, by producing sufficient contrast so that pulsations can be studied. This is done in a simple fashion by producing gas

artificially in the stomach (the patient takes a solution of tartaric acid followed by sodium bicarbonate) and by inflating the colon with air. If pulsations are present at all, they are clearly visible along the borders of the mass. Occasionally these pulsations may be noted within the physiological stomach air-bubble. Pneumoperitoneum is of aid in making the diagnosis.

A film taken with the Potter-Bucky diaphragm technique almost invari-



FIGS 254, 255, 256—59 yrs, M. Dyspnea and slight degree of pain in thoracic spine region for one year. Typical syphilitic aortic regurgitation and tabes. *Ant. view*—moderate enlargement of silhouette caudad and to lt. Diffuse widening of aortic shadow with prominence along course of descending aorta (bl arrow). *Lt. ant. obl. view* fusiform aneurysm of cranial portion of descending thoracic aorta (bl arrows). *Rt. ant. obl. view*. typical erosion of several



vertebral bodies (wh arrows), at level of aneurysm. Postmortem. Lt. ventricular hypertrophy and dilatation. Syphilitic aortitis and atherosclerosis of whole thoracic aorta. Aortic regurgitation. Aneurysm of orange size, arising from dorsal aspect of descending aorta and eroding lt. half of 5-7th vertebral bodies.

ably reveals the presence of lime salt deposits. Their curvilinear arrangement, in parts encircling the round or oval shaped mass, is a characteristic feature. The extent of calcification does not necessarily correspond to the whole surface of the aneurysm. Not infrequently the true aneurysm is encircled by a dissecting hematoma. Calcification in the main branches of the abdominal aorta is often associated. This is seen chiefly in the iliac and hypogastric arteries, occasionally also in the renal, splenic and mesenteric arteries. Erosion of the twelfth rib or of the transverse processes of the vertebral bodies should be

suspected. An erosion along the ventral contours of the vertebrae is easily overlooked or impossible to diagnose with limitation to the anterior view because of the persistence of the typical trabecular structure and because the cortex of the cephalic and caudal laminae is not involved in the process as a rule. Lateral



FIGS 257, 258, 259—65 yrs, M Pain in lower part of spine. Findings of neurosyphilis. Pulsations at rt base posteriorly A, accentuated. B.P. 110/70 Wassermann positive. E.C.G. n.s.r., intraventricular block, deep Q. Aortitis and aneurysm. Ant view: large, sharply defined mass (bl arrow) in middle and lower rt. lung field, outlined by lime salt deposits. Lt border of mass is noted as a dense shadow within cardiac shadow. Marked prominence of aortic knob without appreciable dilatation. Rt ant obl view: (A): cranial portion of descending thoracic aorta (wh arrows) disappears within huge aneurysmal mass, caudal border of which is outlined by lime salt (bl arrow). Rt ant obl view. (B) displacement of esophagus along ventral contour of aneurysm.



views are much more revealing. In this latter position, freed from the overlapping, unhomogeneous shadow of the spine, the calcification is much better visualized and is usually found to be quite extensive and of a fusiform or spherical arrangement. The vertebral bodies may or may not be eroded. If they are, each single bony segment displays a hollow depression, while the upper and lower end plates and the respective intervertebral disc are usually not affected.

A diffusion of irregularly outlined lime salt deposits, not of the shell type arrangement, is noted in the following conditions: teratomatous tumors, in hemorrhagic areas in tumors that may occur in the myomatas and hypernephromatas, tuberculosis of the kidney, and calcification in the mesenteric lymph nodes.

**DISSECTION OF AORTIC WALL** (the so-called dissecting aortic aneurysm). This usually develops on the basis of atherosclerotic and hypertensive disease. In rare instances the size and macroscopic appearance of the aorta is normal while microscopic study reveals degeneration of the media. The size, extension and amount of circular extent vary greatly. Rupture near the aortic arch is common and the dissection may travel, between the layers of the media or between media and adventitia, either distally or proximally. The newly formed blood path may almost completely encircle the aortic tube, thus forming a case or coat. More frequently only half to two-thirds of the circumference is involved. Thus the blood clot may come to lie essentially in a frontal or sagittal plane to the aortic vessel proper. The form may be either saccular-circumscribed or, more often, cylindrical-diffuse.

Once the clinical diagnosis is suspected, the roentgen examination must be made with the patient in bed.

A marked fluid collection in the pleural cavity or a diffuse extravasation of blood into the mediastinal tissues can easily interfere and make the roentgenological diagnosis impossible. With the dissection extending between the brachio-cephalic vessels or almost completely encircling portions or the entire length of the thoracic aorta, the differentiation of a simple dilatation or cylindrical or saccular aneurysm of the thoracic aorta is impossible. An exception to this general statement must be made. The aorta may contain lime salt deposits and its contour will then be visualized as a denser, linear shadow within a larger, usually cylindrical but occasionally globular and otherwise homogeneous shadow mass. If the newly formed aneurysm involves only a part of the circumference of the aortic vessel and has a predominantly lateral location, with respect to the direction of projection, it will be visualized as an outer, lighter shadow, usually of fairly even width, superimposed in a shell-like fashion upon the aortic shadow proper. The latter then appears as a more dense nuclear shadow, the



FIG 260—75 yrs, F Dizzy spells No abdominal complaints Large mass in upper abdomen predominantly on lt. side—not pulsating First clinical impression: malignancy of stomach Systolic murmur over apical and aortic area Normal pulsations in arteries of legs BP 164/84 Wassermann negative ECG n.s.r, lt axis dev Arteriosclerotic and hypertensive cardiovascular disease Lat view. lime salt deposits (wh arrows) outline aneurysmal dilatations of abdominal aorta Spine not eroded



size and shape of which is commonly, though not necessarily, altered by dilatation, fullness and tortuosity. Again, lime salt deposits may form a line of demarcation.

**IN CHILDHOOD.** The incidence is very rare indeed and aneurysms are more



FIG. 261—46 yrs, M Severe lower back pain for 3 days. Heart enlarged, no murmurs,  $A_2$  accentuated. B P 200/130 Leucocytosis Wassermann negative Clinical diagnosis coronary thrombosis *Ant view*: marked enlargement of silhouette to lt, diffuse widening of aortic shadow. Trachea slightly displaced to rt Marked haziness in lung fields Course sudden death during attack of precordial pain. Postmortem. marked lt ventricular hypertrophy and dilatation Atherosclerosis and acute mesoarteritis of aorta Perforation of intima at insertion of origin of ligamentum arteriosum, dissecting, intramural encircling hematoma, reaching from aortic valves to abdominal bifurcation with secondary rupture into lt pleural cavity. Width of aorta-proper normal (5 to 6 cm); thickness of hematoma 4 cm. Chronic glomerulonephritis No evidence of coronary thrombosis The widening of the aortic shadow was caused by an encircling hematoma; this was added to the shadow of the aorta and could not be differentiated from it

glomerulo-nephritis or malignant hypertension; occasionally 3) rheumatic; 4) syphilitic (further studies are needed), and 5) coarctation of the aorta.

Because of the high distensibility at this age, the aorta may undergo a considerable stretching during life (dynamic dilatation) as a sequel to the high pulse pressure in the presence of aortic regurgitation and high blood pressure, both systolic and diastolic. The roentgenological appearance may well be one of a diffuse cylindrical dilatation of a greater degree. Fluoroscopic observation and films taken at both the systolic and diastolic phases reveal considerable diminution during the latter phase in cases of aortic regurgitation. But it is always surprising to see, at the postmortem examination, how small the aorta actually is when the distending force has ceased to exist.

**DIFFERENTIAL DIAGNOSIS.** It will be assumed for the following discussion of conditions to be differentiated that the individual under study is the bearer of a normal aorta. It is furthermore assumed that the study of the different views and, if necessary, the use of a special technique (over-exposure; Potter-

Bucky diaphragm), will permit the visualization of at least a portion of the aorta. This vessel, in its ascending and/or transverse portion may be displaced but is assumed not to reveal, in its visible portions, fundamental

A substernal goiter may consist of intrathoracic extensions of one or both lobes of the thyroid. The shadow is triangular in shape with convex borders, and the apex points caudad. If the patient is examined in various views, at least part of the normal appearing aortic arch can be visualized. Unless there is malignant degeneration of the thyroid gland with infiltration into the surrounding tissues, the gland moves during swallowing and coughing. The trachea usually reveals some degree of displacement and narrowing. This is observed in the region of the neck.

Tumors of the thymic region are located in the anterior mediastinum at each side of the median line and usually extend to the base of the heart. The main axis is perpendicular to the long axis of the body. The delineation may be either sharp or, in case of invasion into the surroundings, hazy.

Certain benign mediastinal tumors, often oval in shape, perfectly smooth in outline, and, as a rule, of a homogeneous density, show their main axis in the direction of the long axis of the body. Here belong teratomas, hydatid cysts and neurogenic tumors, such as neurofibromas and ganglioneuromas. Teratomas may reveal some area of calcification but none of these tumors show an encircling distribution of lime salts. The neurofibroma is typically located in the posterior mediastinum and associated bone changes are common, for example, widening of the intervertebral foramina. The retropleural site of these tumors may be demonstrated by means of a diagnostic pneumothorax.

Tumor-like involvements of the mediastinal lymph nodes (sarcoma, lymphogranuloma, leukemia) are usually bilateral and most often show polycyclic borders which sometimes are not sharply delineated and often extend without sharp distinction into the soft tissue shadows of the neck. Uniformly growing, unisentric tumors which extend from the supraclavicular region toward the base of the heart have an almost straight outline. Lack of homogeneity is noted when the constituent parts of the tumors have different size and depth. There is generally no displacement of either trachea or esophagus. These growths will often encircle the aorta and this results in expansile pulsations along their borders. Radiation therapy leads, in many instances, to considerable regression in the size. With the polycyclic type, the outline becomes straightened and a column-like widening of the upper mediastinal shadow results. In other instances complete disappearance of the shadow mass is noted which may be permanent or transient.

Malignant bronchial tumors of the hilar region invade the surrounding lung tissue in a radiating fashion. If large, they may produce marked signs of bronchial stenosis.

Mediastinal pleurisy is rare. It has either a ventral or dorsal site and extends in the form of a paramediastinal band or crescent-like shadow.

A unilateral sacculated pericarditis may have either a smooth semicircular or a polygonal outline. Pulsations can be present because of transmission. In no view can it be separated from the shadow of the aorta, and other roentgenological findings for adhesive pericardial disease should be postulated, such as absence of pulsations along the cardiac borders. Otherwise the differential diagnosis rests upon other methods of study.

An aneurysm of the left aorta extending into the right . . .

views reveal an exclusively dorsal site with a typical displacement of the esophagus and also a widening of the angle of bifurcation. The lung fields disclose chronic congestion.

An aneurysm of the pulmonary artery tends to develop craniad and dorsad and this is usually associated with a marked dilatation of its main intrapulmonary branches and increased pulsations. The esophagus may exhibit a marked impression below the level of the aortic arch. A pouch at the left anterior circumference of the aorta develops craniad and ventrad (direction of the blood stream), but changes in the pulmonary artery branches are not observed unless the aortic aneurysm has ruptured into the pulmonary artery. Roentgenological as well as clinical signs of right ventricular enlargement are observed whenever an aortic aneurysm occasions pulmonary artery stenosis.

Diseases of the esophagus, such as cancer, diverticulum, and general dilatation subsequent to cardiospasm, are easily distinguished by examination with barium.

Tumors of the chest wall often reveal their origin from a rib or from the spine. They do not arise with a broad base from the mediastinal shadow. A diagnostic pneumothorax is of help provided the tumor has not already extensively invaded the pleura. The more rapidly growing types respond well to irradiation.

The fusiform shadow of a cold abscess of the spine causes no pulsations along the adjacent contour of the barium-filled esophagus. A careful study of the spine usually reveals some changes such as narrowing of an intervertebral disc space or structural changes in one or several vertebral bodies. Only a very superficial examiner will confuse the scoliotic spine with an aneurysm.

The findings of metastatic changes in the lung fields and in the bony skeleton will sometimes disclose the clue for differential diagnosis.

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## CHAPTER X

### DISEASES OF THE PULMONARY ARTERY AND ITS BRANCHES

As compared with the systemic circulation, the pulmonary circulation shows certain distinguishing features. Its diseases are of more manifold etiology and the different age groups are affected almost equally. The whole pulmonary circulation is crowded within a much smaller area and is visible by roentgen rays almost in its entirety.

**Etiology and Anatomical Forms.** Very different etiological factors occasion the same or a similar macroscopical aspect of the pulmonary artery and its branches. The smallest vessels are found to be most severely affected by two conditions. One is a productive arteriolosclerosis; the second an end- or panarteritis. Both may be associated with widening, atheroma and calcification in the larger branches and trunk. In both thrombotic occlusions may occur. The second type is found especially in combination with a destructive-sclerosing broncho-pneumopathy, some of these cases are of syphilitic origin; in others, inhalation of intoxicating war gases plays a rôle. Syphilis may also affect the trunk, much like in mesaortitis, and is sometimes combined with it. Congenital malformations are commonly responsible for dilatation of the pulmonary arterial trunk and occasionally of the branches also. Nearly half the aneurysms of the pulmonary artery are associated with congenital cardiovascular malformations such as interatrial septal defect, patency of the ductus arteriosus, stenosis of the pulmonary orifice. Approximately half of all cases with thromboendarteritis, occasionally complicated by mycotic aneurysms, are found to be associated with patency of the ductus arteriosus. A congenital hypoplasia of the wall (deficiency of the muscular or the elastic layers or both) has been found in a number of instances. In certain types of transposition, the dilated pulmonary trunk is hidden behind the ascending aorta. Other aneurysms, which may be single or multiple, cylindrical or saccular, and which are most likely congenital in origin, are located in the periphery. There has been observed enlargement of the pulmonary artery and its branches not associated with other congenital cardiovascular malformations nor with arteriolosclerosis. The etiology of these "idiopathic" cases is obscure. An abnormal division of the truncus arteriosus communis has been conjectured. Dilatation is likewise found in the presence of an acquired regurgitation (and stenosis) of the pulmonary orifice. Traumatic rupture of one of the larger branches leads to a paramediastinal sac-like formation. Here we may mention the perforation of the aorta into the pulmonary artery and the formation of a dissecting aneurysm. Some widening of the pulmonary artery and atherosclerotic processes in the lesser circulation are found in marked mitral valvular disease. A rheumatic endarteritis may sometimes account for these findings. The thinness of the wall, the rich content of connective tissue (loose collagen) and the lack of resistance along its anterior and lateral aspect make the pulmonary artery more apt to undergo changes than the ascending aorta. Athero- and arteriosclerotic changes with narrowing of the smaller vessels and widening of the larger ves-

sels and the trunk may be finally found in the course of fibrosis of the lungs and anoxemia if the following conditions prevail extensively: bronchiectasis, emphysema with chronic bronchitis, chronic pneumonia, pneumoconiosis. Infection probably plays the most significant rôle. Adhesive pleural disease may also be a contributing factor. On the other hand emphysema that is found to be associated with an obliterative vascular process need not always be considered as a causal factor. It may be merely a coordinated feature. When using the term "pulmonary sclerosis," an attempt should be made to specify the primary, i.e., arteriologenic type or the secondary, i.e., the cardiogenic or pulmogenic type.

The lungs may reveal parenchymatous changes of various types (see



FIGS 262, 263—55 yrs, F. Moderate cyanosis, marked cardiac enlargement, hypertension  $P_2$  accentuated. *Ant view*, marked enlargement of cardiac silhouette with prominent pulmonary arch and marked widening of pulmonary artery branches. *Rt ant obl view*, the barium-filled esophagus reveals 2 impressions, the cranial (wh arrow), at level of aortic arch, is physiological, the caudal (bl arrow) is caused by an impression of right pulmonary artery branch in connection with displaced lobe of bronchus. Pulmonary artery dilatation with impression on esophagus.

above). Congestion may be present but it is interesting that in some instances of primary arteriosclerosis a paucity in the blood content of the lungs has been mentioned.

Extensive, occlusive processes in the smaller vessels may be found with a practically normal appearance in the larger vessels and trunk, but far more often there is concomitant enlargement. The dilatation, even aneurysmal in size, may be limited to the trunk or to one of the branches only. Bronchopulmonary sclerosis as well as intrinsic disease may lead to constriction of the branches. Displacement and widening of the pulmonary artery and displacement with kinking of its branches is noted in the course of sclerosing cirrhotic-cavitary tuberculosis of the left anterior (upper) lobe of the lung.

Calcification is relatively rare. It may affect the pulmonary trunk only,

especially in the presence of an acquired aneurysm, or all the lung vessels extensively.

Paresis of the left recurrent laryngeal nerve and bronchial stenosis are occasionally observed in connection with aneurysmal dilatation of the pulmonary artery. Dyspnea, cyanosis and hemoptysis are commonly associated with many types of pulmonary arterial disease and the erroneous clinical diag-



FIG. 264—34 yrs, M. Several attacks of rheumatic fever since age 22. Failure for 2 years. Mitral stenosis, aortic stenosis and regurgitation. B.P. 100/60. E.C.G.: n.s.r., rt axis dev. Ant. view: silhouette moderately enlarged with prominence of second pulmonary arc at lt. Pulmonary artery and pulmonary artery branches of increased density; rt main lower branch 2.0 cm in size. Aortic knob clearly visible. Fluoroscopy: second and third lt. arch pulsate alternately. Very slightly pulsations of hilar vessels. Rt ant. obl. view (illustration omitted) prominence of pulmonary conus ventrad, lt atrium enlarged dorsally. Course:

lesion with secondary dilatation and atherosclerosis of pulmonary artery system and aortic knob was clearly visible because of presence of aortic valvular lesion. The intracardiac calcification was not diagnosed during life.

FIG. 265—47 yrs, F. History insignificant. For last few years precordial oppression and mod. rt aus. enormous prominence in region of pulmonary conus and artery (bl. arrow), enormous enlargement of all intrapulmonary branches as noted in longitudinal and cross sections (wh. arrows).

nosis of emphysema or congenital cardiovascular disease has been made far too frequently.

The right heart is the site of hypertrophy and dilatation and a marked prominence of the conus region is regularly found.

Roentgenologic Findings. STATIC ASPECTS. It must be understood that

an unusual prominence of the pulmonic arch or branches does not necessarily occur even though vascular disease may exist either in the periphery or in the central region.

No convincing roentgenologic evidence has been given so far for pulmonary artery branch thrombosis or embolism; nor has there been roentgenologic study in man of the dilatation of the pulmonary artery and right heart chambers which is believed to occur subsequent to massive pulmonary embolism (acute cor pulmonale). Instructive experimental studies with respect to pulmonary embolism have been carried out on dogs, and should be mentioned

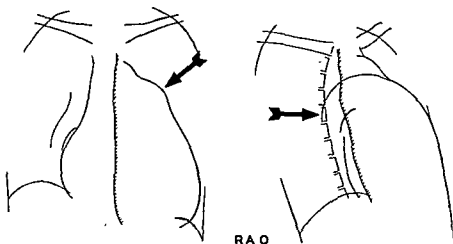
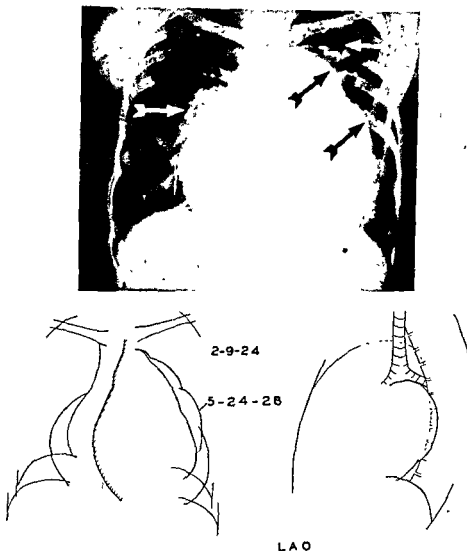


FIG. 266—31 yrs, F Increasing dyspnea for several years. Marked weakness and ankle edema; somnolence, moderate cyanosis, bronchiectasis, liver enlargement, marked cardiac enlargement.  $P_2$  acc. Tachycardia. BP 90/70. ECG: n.s.r., right axis dev.;  $T_1$  negative. Wassermann negative. *Ant view*: large heart; L 16.6, B 14.1, T 15.3, Th 22.3 cm. Marked prominence of pulmonary conus and pulmonic artery (bl arrow). Aortic knob small but visible. Central arterial pulmonary branches enlarged, of increased density. Main lower branch 1.8 cm. Pulsations not increased. Lung fields clear. *Rt. ant obl view*: mass of heart in front. Lt. atrium not enlarged. A dense band-like shadow is noted corresponding to course of pulmonary artery and main rt branch (bl arrow). Postmortem: marked hypertrophy and dilatation of right heart chambers. Left ventricle not visible in situ. Enlargement of pulmonary artery and of its branches. Purulent and obliterative bronchitis. Extensive athero- and arteriosclerosis of the entire pulmonary arterial system. Canalized thrombi in many branches.

here. The most suitable type of artificial embolism is obtained by filling 30 to 40 cm.-long pieces of rabbit's intestine with clotted blood and adding lipiodol for roentgen visualization. When this type of artificial embolus is introduced into the venous circulation of dogs and reaches the pulmonary artery, it will curl up and buckle like the emboli observed in human pathology. These artificial emboli were introduced into the venous current. Roentgenograms were done and postmortem studies carried out. Instant death took place only when the trunk or both main branches were plugged. Obstruction of one branch exclusively produced death only once, and that after more than



FIGS 267, 268—19 yrs, M. Rheumatic fever at age 14 followed by mitral endocarditis and cardiac failure. Patient active in athletics following recovery. At age 18½ marked cardiac enlargement, precordial bulge, mitral and tricuspid regurgitation  $P_2$  much accentuated ECG: auricular fibrillation, rt axis dev.,  $T_2$  diphasic. Rapidly progressing failure, without rheumatic activity. *Ant. view* (age 14): marked enlargement, mitral configuration; L 15.3, B 11.4, T 15, Th 23.2 cm. Moderate prominence of pulmonic arch. Rt ant obl view (illustration omitted) marked enlargement of lt atrium. *Ant. view* (age 18½, orthodiagram and film) very large silhouette, L 17.8, B 14.6 (max. 16.9), T 18.5, Th 24.6 cm. Rt. lower cardiac contour markedly curved. Pulmonic arch greatly prominent (upper bl. arrow), caudad a third prominence is noted, intersecting with lt lower contour (lower bl. arrow). Intrapulmonary branches are moderately dilated (wh arrows). Absence of congestive lung failure. Esophagus displaced to rt. Fluoroscopy reveals absence of pulsations along third lt arch. Rt ant obl view (illustration omitted): second lt. arch remains ventral while third disappears; esophagus displaced dorsad. *Lt ant. obl view*: Corresponding to level of third arch, a mass overlaps spine and displaces lt. bronchus cranial. The third arch, therefore, corresponds to lt atrium which developed, in an unusual manner, pre-

two weeks had elapsed. This is an interesting corollary to the clinical experience that rapid death due to pulmonary embolism is caused by complete obstruction of the trunk of the pulmonary artery or by simultaneous obstruction of both its branches in 70% of all the cases while in the remaining 30% emboli more peripherally located seem to set up a fatal neuro-vegetative reflex.

Usually an increase is seen in the length and in the lateral projection of the middle arch, together with a marked degree of density. This prominence encroaches upon the retrosternal space in the right anterior oblique view, and upon the ventral and caudal aspect of the window of the aorta in the left anterior oblique view. In the former view, a knob-like prominence into the posterior mediastinum below the level of the aortic arch and above the level of the left atrium is commonly present. This represents the right main branch of the pulmonary artery, coursing horizontally from the left to right, either actually enlarged or displaced dorsad. At this level there is usually a resulting exaggerated indentation of the esophagus either in the form of a curve distinctly separate from the cranially situated aortic arch impression or blending with it. There is discussion as to whether this indentation is due to a direct impression by the enlarged pulmonary artery branch or whether this vessel displaces the left bronchus, and to a small extent the lymph nodes at the bifurcation of the trachea, against the esophagus. The latter supposition is anatomically more probable. The marked prominence of the pulmonary artery and conus may, in exceptional instances, be distinguished exclusively in the right anterior oblique view, as when an aneurysmal pouch develops to the right in front of the aorta and the right auricular appendage. In the presence of transposition of the great vessels, the pulmonary artery is situated behind the aorta, and then the enlargement of the trunk cannot be discerned though the dilated branches may be clearly defined in each hilar region. A considerable extension of the shadow of the descending aorta into the left lung field easily leads one, with limitation to the anterior view, to err in estimating the degree of prominence of the middle arch. The aortic knob usually remains clearly visible cranially to the middle arch contour but two exceptions must be noted: one is the presence of mitral valvular disease (especially high grade mitral stenosis), the other occurs with congenital anomalies in position and size of the aorta.

Dilatation of the branches is noted by an unusually good visibility of the entire central and peripheral vascular pattern; this is enhanced by the customary absence of congested lung fields, exceptions are mitral valvular disease, or left ventricular failure. *The large hilar branches must be studied with some*

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dominantly to it Postmortem: Marked hypertrophy and enlargement of all four heart chambers. Second lt arch corresponds to pulmonary artery, third to lt atrium. Mitral and tricuspid regurgitation. Size of pulmonary artery 10 cm., aorta 5.5 cm. Fibrosis of heart muscle. Arterio- and arteriosclerosis of pulmonary artery circulation. The absence of congestive lung failure should be related to the tricuspid valvular lesion as well as to the vascular sclerosis in the lesser circulation. A systolic distension of the lt. atrial contour was not observed in spite of the presence of mitral valvular regurgitation. The position of the diaphragm was low, presumably due to the marked increase in heart size and weight.



angle of rotation because of the interference of the cardiac and pulmonary conus shadow in the straight anterior view. The comma-like descending branch on the right is most impressive in its massive appearance and so are the dense, disc-like cross sectional areas of the vessels. The right anterior oblique view reveals a considerable decrease in the transparency of the retrocardiac space because of the projection of all vessel shadows into it. At first glance, an enlargement of the left atrium may be diagnosed. A more careful analysis, however, reveals the unhomogeneous character of this shadow and subsequent

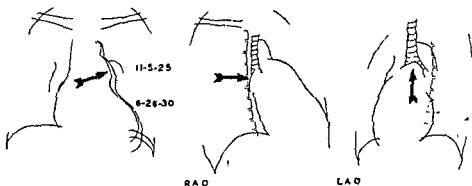


FIG 269—39 yrs, F Septic endocarditis at age 21, followed by development of valvular lesion. Recurrence of endocarditis at age 32. Slight degree of cyanosis; dullness, pulsations, systolic-diastolic thrill and murmur at 2nd and 3rd R intercostal spaces B.P. 105/70 E.C.G.: n.r., rt. axis dev. Pulmonic regurgitation. *Ant view*: Cardiac silhouette enlarged downward and to lt.

	L	B	T	Th
1925	14.9	9.4	13.5	23.8
1930	16.4	10.2	14.2	23.8

Aortic knob normal appearance. Marked prominence pulmonic arch (bl arrow). Main pulmonary artery branches dilated, main rt lower branch 1.8 cm in diameter. Lung fields clear. Fluoroscopy reveals pulsatory amplitude at pulmonic arch, 0.8 cm, cranial portion of lt cardiac contour 0.5 cm, and main hilar vessels 0.5 cm. The density of hilar vessels changes between systole and diastole. Pulsations at lt cranial cardiac contour (rt ventricle) and pulmonic arch alternate in direction—see-saw movement. *Rt. ant obl view*: marked prominence of pulmonary conus and artery with knob formation in posterior mediastinum (bl arrow), lt atrium not enlarged. *Lt ant. obl view*: aortic window filled out by a dense pulsating shadow (bl arrow). Right ventricular enlargement may occasion an extensive widening of the silhouette to the lt. Hilar pulsations in the presence of pulmonic regurgitation correspond to the waterhammer radial pulse in the presence of aortic regurgitation.

examination of the course of the esophagus, by means of barium paste, shows no appreciable displacement at the level of the left atrium.

One may differentiate, from a descriptive viewpoint, those cases where dilatation is limited to the trunk, from those where both trunk and branches are dilated. The advantage from a diagnostic viewpoint is limited. In the former group belong the mechanical displacements; certain cases with stenosis of the pulmonary orifice or at the bifurcation, and beriberi and thyrotoxic disease of a higher degree. In the latter group may be mentioned regurgitation of the pulmonic valves, mitral valvular disease, interatrial septal defect, patency

of the ductus arteriosus, and numerous instances of degenerative and infectious disease (arteriosclerosis, syphilis).

Isolated dilatation of one or a few of the branches, traumatic or congenital in origin, shows saccular or cylindrical shadow formation, sometimes far out in the periphery of the lung fields. Phlebectasies and arterio-venous fistulas have been observed. The latter seem to cause polycythemia. A mottled area in a lung field may represent an angioma. In one such case observation over a number of years showed progression in size and density, and the postmortem



FIGS 270, 271—8 yrs, F. Known to have congenital heart disease. Height 112 cm, weight 16.5 kg. Precordial bulge. Marked cyanosis and clubbing. Marked thrill and rough systolic murmur all over heart. ECG: nst, rt axis dev. Ant view: silhouette moderately enlarged, with prominence of pulmonic arch. Aortic shadow not visible. Enormous enlargement of all branches of pulmonary artery. The latter appear partly as band-like streamers (longitudinal section) or as round dense areas (cross section). Fluoroscopy reveals expansile pulsations of all these shadows. Rt ant obl view: Lt atrium not enlarged. Prominence of pulmonic knob into posterior mediastinum (bl arrow). Congenital cardiovascular malformation with marked enlargement of pulmonary artery and branches. The diagnosis of bilateral infiltrative process should be avoided.

examination revealed angiomas in other organs also, hence a mesodermal dystrophy was present.

Calcification is best noted in the oblique views as a curvilinear, dense shadow, either along the cranial contour of the pulmonary artery or outlining the shadow areas which represent the cross sections of the two main branches. Presence of lime salts accounts, in part, for the high degree of density which often is noted in the roentgenograms depicting dilatation of the pulmonary artery and its branches.

The heart reveals the characteristic findings of right sided enlargement except in the group with mechanical traction cranial and to the left. Low position of the diaphragm, either as an expression of body build or the result of emphy-

sema tends to be associated with a considerable prominence of the silhouette to the right. With the stouter type of individual, prominence to the left prevails; here one also finds a well-pronounced junction between the pulmonary conus prominence and the left caudal portion of the silhouette.

The marked prominence of the pulmonary conus and artery region or the

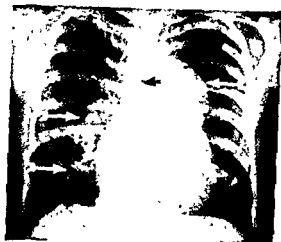


FIG 272 — 17 yrs, M. No history of infectious disease. Cardiac lesion diagnosed in childhood. Full compensation. Systolic lifting of precordium; dullness, systolic pulsations and thrill and systolic-diastolic murmur in 2nd lt. intercostal space. B.P. 135/80. ECG: n.s.r., rt axis dev., T<sub>1</sub> s.s. diphasic. Rbc 5.4 mill., hb 105%. Wassermann neg. Ant view. globular shape of silhouette. Aneurysmal dilatation of pulmonary artery and conus (lt. wh arrow), displacing esophagus to rt (short bl arrow). Dilatation of pulmonary artery branches (rt. wh arrows), rt main branch measures 2.1 cm. Fluoroscopy: expansile pulsations of pulmonary trunk and branches. Absence of congestive failure. Rt ant obl view (illustration omitted): prominence of conus ventrally and prominence of pulmonary knob into posterior mediastinum, causing displacement of esophagus dorsad. Lt atrium not enlarged. Congenital cardiovascular malformation with pulmonic regurgitation, aneurysmal enlargement of pulmonary artery and rt ventricular predominance.

large shadows of the main branches have repeatedly and erroneously been interpreted as mediastinal tumor. The unduly outstanding vessel shadows in the periphery were designated as strands of infiltration; and localized saccular aneurysmal dilatation in the periphery has been interpreted as metastatic malignancy, benign neoplasm (for instance, chondroma) and solitary hydatid cyst.

**DYNAMIC ASPECTS** Pulsations are easily visible and their visualization is facilitated by the contrast between the clear lung fields (exceptions see above) and the dense vascular shadows. A diminution or even absence of pulsation is found in the presence of extensive thrombosis which is not uncommonly encountered in aneurysm. It is, therefore, not justifiable to reject the vascular origin of a mass in question because of the absence of pulsation. One must distinguish between transmitted and intrinsic pulsations; the former reveal changes neither in volume nor in density during a cardiac cycle. They are observed bilaterally with an exaggerated and tachycardic heart action or limited to the right hilar

to the aforementioned wall changes that are probably often combined with a hypertensive state in the lesser circulation. Healthy children also occasionally

show this sign. No definite pulsations were noted in those cases where in the presence of mitral valvular disease a relative pulmonic insufficiency was thought to be present. Where well-marked pulsations of the pulmonic arch are noted, the left cardiac contour also shows pulsations of about equal size but alternating in direction. Thus a kind of a see-saw action is observed. The amplitude of the aortic excursions is comparatively small. This disproportion between aortic and cardiac amplitude obviously proves that the whole left lower contour is formed by the right ventricle with the left ventricle rotated dorsad. Expansile pulsations in the hilar region were originally thought to be an expression of a venous pulse, consequent upon a reflux of blood into the pulmonary veins in the case of mitral regurgitation. Later on their arterial character was recognized and believed to be characteristic of pulmonary artery sclerosis. Actually they are found in conditions associated with an increased pulse pressure in the lesser circulation and they are most typically observed in the presence of regurgitation of the pulmonic valves. In this instance an actual collapsing pulse is noted along the contours of the pulmonary trunk and branches with alternation in density between systole and diastole, and even a difference in the transparency of the peripheral lung fields may be occasionally observed. The pulsations of the lung vessels are the equivalent of the collapsing pulse in the radial arteries in the presence of aortic valvular regurgitation.

For further discussion see also chapter XII.

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#### CHAPTER X DISEASES OF THE PULMONARY ARTERY AND ITS BRANCHES

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## CHAPTER XI

### PERICARDIAL DISEASE

**Pericardial Effusion.** The roentgenological diagnosis of fluid within the pericardial cavity is possible only if certain changes occur in the size and shape of the cardiovascular silhouette and in the pulsations along its borders. No information is obtained as to the etiology or type of the fluid. A moderate amount of fluid can quickly accumulate because the surface of the pericardium is greater than that of the heart. The pericardium can be distended to a wide sac provided this process of distention is relatively slow. A rapid distention, within the course of a few hours is not possible, and cardiac tamponade results from a sufficient degree of intrapericardial pressure. The accumulating fluid, if not loculated, obeys the law of gravity, and changes along the silhouette are found when altering the position of the body, particularly when the pericardial sac is not stretched to its maximum.

It is assumed for the following discussion that the pericardial cavity is free from either a partial or total obliterating process.

The amount of fluid which will cause positive roentgen signs to appear is not exactly known. This, in part, depends upon the size of the heart. 250 cm<sup>3</sup>. in the adult is perhaps the minimal amount while as little as 10 cm<sup>3</sup>. has been diagnosed in infants. The distribution of fluid seems to depend, in part, upon the prevailing body position.

**Silhouette.** A collection of small amounts of fluid should be suspected first when the waist of the silhouette is straightened. A real bulge is never noted unless encapsulation has taken place. Serial studies may have to be undertaken to find the significant sign of an appearance or disappearance of such a deformation along the silhouette border. Secondly, a collection of fluid should be looked for in the region of the inferior posterior recess or sinus of the pericardial cavity which is approximately at the level of the inferior vena cava. While this vessel in the lateral view is characterized normally by a straight or slightly concave dorsal contour, a convex bulge is noted when the aforementioned sinus is distended by fluid. Observation of this sinus contour is interfered with by the presence of a pleural effusion. A good sized effusion fills up all available sinuses and as a result of the extension of fluid into the superior anterior recess, the vascular shadow is shortened. This becomes more pronounced in the recumbent position. The degree of shortening depends upon individual variations of the cranial pericardial reflection line. The transverse diameter of the vascular shadow rarely increases to any great degree. This is due to the fact that the accumulation of fluid takes place predominantly ventrad to the vessels. A reduction to a short pedicle is not observed in those instances where the superior recess is obliterated because of previous disease.

As the fluid accumulates, the

shortening of the vascular shadow is finally present in both recumbent and



upright positions. The collection of fluid between the mass of the heart and the diaphragm displaces the liver and may displace and deform the stomach air bubble. The appearance of an effusion of high degree is thus characterized by a short, relatively small pedicle and by a large silhouette with a circular smooth outline at either side which emerges from the vascular shadow almost at a right angle. T prevails over L. Contrast in the left supradiaphragmatic area diminishes considerably, mainly because adjacent portions of the lung become atelectatic. The pericardio-diaphragmatic angle at the right is quite acute and small and this explains why percussion does not reveal the presence of lung tissue between the mass of the effusion above and the liver below. An obtuse angle does not result because the pericardium does not distend as much along its line of fixation as it does cranially. The left lateral view demonstrates that the shadow reaches as far as the posterior chest wall. A clear outline of the silhouette is not obtained, however, because with this degree of distention the air content of the lungs is diminished.

It is often believed that individuals who present this roentgenologic picture must be very ill and consequently bedridden. This is not necessarily so and patients with a large pericardial effusion of tuberculous origin may indeed be up and about for some time.

Immediately following removal of fluid by paracentesis, provided that air has not entered, the silhouette appears less rounded, the caudal portions are wider than the middle area and a trapezoid or triangular silhouette results. Examination in a recumbent lateral position reveals a considerable displacement of the silhouette shadow.

Small amounts of exudate which occur in the course of rheumatic heart

transudates in the  
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this etiology, however, may appear in the course of a few days, appreciably alter the silhouette and be resorbed rapidly. On the other hand, effusions of tuberculous origin, particularly under the therapeutic influence of salicylates, may persist for many months. Transudates in cases of cardiac failure may be resorbed rather rapidly, the silhouette correspondingly diminishing. This is often interpreted erroneously as an actual diminution of the heart size itself.

Injections of lipiodol have been made into the effusion with the idea of locating pockets of adhesions. It is seen to settle first in drops along the caudal aspect in the form of a crescent. Later, it aggregates in a few large collections.

**Pulsations.** They cease first along the caudal border. This sign is best studied after artificially increasing the gas content in the fundus of the stomach. Pulsations along the other borders of the silhouette first lose their alternating character and are later replaced by a diffuse undulation. The amplitudes of these transmitted pulsations depend upon the vigor of the heart action. They are, for instance, quite considerable in the presence of an acute infectious pericarditis in childhood and in association with an aortic or mitral regurgitation, provided the myocardial contractions are strong. Pulsations may cease entirely. Provided that there is no compression atelectasis of the

left lower lobe, minimal pulsations may become quite noticeable if the patient is bent over on his left side.

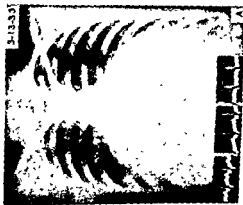
The clinical apical thrust may be felt well within the silhouette, and such a finding favors the diagnosis of an effusion.

If fluid accumulates quickly, cardiac tamponade will result as soon as there is a relatively small amount of fluid, i.e., 250 cm<sup>3</sup>. This is seen with a sudden hemorrhage. The shape of the silhouette does not change considerably and if an aortic configuration were present it would persist. Thus the absence of pulsations, without much enlargement, is an important diagnostic sign. This sign may decide in favor of an operation if a heart wound is suspected. On the other hand, the presence of normal pulsations may spare the patient an operation. The silhouette, however, will pulsate in spite of a bleeding heart wound if blood is draining from the pericardial into the pleural cavity. In this case as well as with a pleuropulmonary hemorrhage, the mediastinal silhouette soon becomes poorly visualized. Fluoroscopy is a very important procedure whenever a wound of the heart is suspected but it must be performed very carefully because of the danger of the collapse of the patient.

*Lung Fields.* The appearance of the lung fields may vary in the presence of a large pericardial effusion. Normal transparency is observed with an inflammatory etiology, i.e., rheumatic or bacterial infection. The accumulation of fluid may result in compression of the right atrium, of the intrapericardial portions of the vv. cavae, and of the hepatic veins, all of which tends to check the blood flow to the lungs. Normal transparency is likewise observed with a pericardial effusion due to severe hypothyroidism. When an effusion develops in the course of left heart failure, i.e., due to hypertension or aortic valvular disease, haziness of the lung fields is observed. Renal failure may be associated with interstitial pulmonary edema that is chiefly of central location and of cloudy-mottled appearance.

*Differential Diagnosis.* It is not always possible, by any means, to differentiate cardiac dilatation from pericardial effusion. The two conditions may well be combined. As helpful signs in favor of pericardial effusion the following may be mentioned: the bulge of the inferior posterior recess; the foreshortening of the vascular shadow, especially in the recumbent position, a considerable shift of the shadow mass toward the dependent side in a lateral prone position; and disappearance of arcs resulting in a rounding out of the borders. If the silhouette is very large there should be added prevalence of T over L; absence or only slight degree of congestive lung failure, and absence of pulsations. Indistinctness and absence of pulsations are also noted in large hearts, but, unless conditions of the chest wall or lungs interfere, clinical evidence of pulsations of the chest wall as revealed by inspection and palpation is usually available.

The heart shadow is not visible within the larger silhouette of the effusion in the form of a nuclear, central denser area. This has been proven by adequate experiments. A central shadow with a uni- or bilateral borderline may be occasioned by a number of conditions, all of which may well be associated with a large silhouette either due to a pericardial effusion or to cardiac enlargement. These conditions are: 1) a large left atrium casting its shadow through; 2) the right atrium visualized within an aneurysmal dilatation of



Figs 273, 274, 275, 276, 277 — 22 yrs. M. Fever, chills, weakness, dyspnea and slight precordial pain for several weeks. Marked increase in cardiac dullness. Apical thrust difficult to discern. Friction rub. Pulse thready, tachycardia, B.P. 100/80. Temperature 100 ECG n.s.r., voltage diminished, T, flat positive, T, isoelectric. Pericarditis with effusion. Ant view (A) — typical high grade globular enlargement of silhouette. Vascular shadow tapped by epigastric route, 760 cm<sup>3</sup> fluid removed and 300 cm<sup>3</sup> air injected. Guinea pig inoculation positive. Typical succussion splash and shifting tympanism. Ant view (B) — pericardium enormously distended and thickened (wh arrow), air and fluid in pericardial cavity, fluid with horizontal level (bl arrow). Lat view (C) — upper recess of pericardial cavity (wh arrow), horizontal fluid level (bl arrow), lat. view (dorsal decubitus) (D) — note sternum, thickened pericardium (wh arrow), air, ant. portion of heart, horizontal fluid level (bl arrow). Course. rapidly accumulating fluid necessitated repeated paracentesis. Eight months later. low voltage, T, isoelectric, T, negative. Ant view (E) — silhouette diminished in size; right cardiac contour fades into diffuse area of pleural thickening. Pulsatory amplitude hardly visible. Course pericardectomy. Death. Postmortem tuberculous pericarditis with synechiae easily disruptible. Pericardium 0.7 cm., epicardium 0.4 cm. in thickness. Thickened fibrous right pleura.



the left atrium; 3) an aneurysm of the descending aorta; 4) a tuberculous abscess of the spine; 5) a paraesophageal diaphragmatic hernia; 6) a marked degree of dilatation of the esophagus in the course of a cardiospasm; 7) a mediastinal pleural effusion. It is noted that all these formations are in a plane other than the mass of the heart plus the encircling effusion. There is one condition to be added: that is, 8) a localized thickening of the pericardium.

**Pneumopericardium.** The presence of air in the pericardial cavity is characterized by three roentgenological findings. There is an area of increased transparency around the cardiovascular shadow and the lung structure is markedly diminished or almost absent. This area of transparency is outlined along its periphery by a dense, ligamentous, curved shadow formation. An unusual activity of the heart shadow is noted. A basal horizontal fluid level is commonly observed which is continuously in motion. The appearance is fundamentally similar whatever may cause the entrance of air. It may be due to a traumatic communication with any of the air-containing adjacent organs, or directly from without, i.e., following a wound or surgical procedure. It may be created involuntarily, in the course of needling for pneumothorax; or purposely, in the replacement of air for pericardial fluid which was removed by paracentesis.

The size of the transparent area depends upon the amount of air and upon the size of the pericardial cavity previous to the entrance or introduction of air. It may be very small in size or it may, at the left side, almost reach the left thoracic wall. In the lateral view it is seen in front to separate the heart shadow from the sternal shadow, while dorsad it may extend to the spine. It extends cranially along the vascular shadow and can be made to outline the caudal border of the heart shadow in the Trendelenburg position. Rarely is the left diaphragm seen depressed in a concave fashion. Resorption of even smaller amounts of air takes at least a few days.

The shadow of the distended pericardium together with the adjacent mediastinal pleura measures several mm. in thickness and increases considerably in the case of productive disease, such as tuberculosis. It extends from the diaphragmatic surface up to the vessel shadow. The height depends upon variations in the anatomic reflection line and upon the degree of distention of the superior recess. The latter may be obliterated and more of the vascular pedicle will then remain visible outside the air-filled pericardial cavity.

The activity of the heart shadow is most remarkable because the presence of air has freed the heart from the influence of the elastic traction of the normal lungs which acts as a rein on the cardiac pulsations. The amplitude is considerably increased. It was determined kymographically in one case to be as great as 1.7 cm. Both systolic and diastolic movements seem to occur much faster than normally. Finally the heart mass swings and rotates considerably and shows hammer-like vertical movements. The kind of changes which the movements of the atrioventricular septum simultaneously undergo remains to be determined under high pressure. The intra-pericardial pressure in pneumopericardium. It was shown that with higher pressures the changes in pressure produced by systole and diastole were less marked, and that in order to

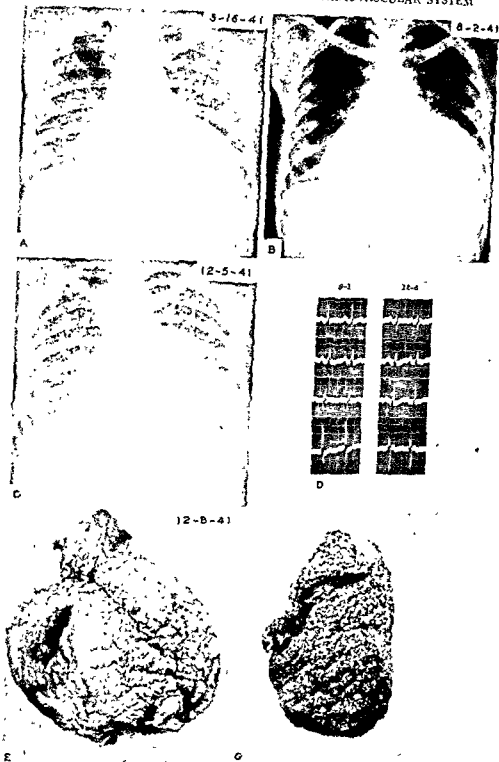


FIG 278, 279, 280, 281, 282, 283, 284—F, 50 yrs History: 10 yrs prior tuberculous peritonitis Progressive weight loss and dyspnea for past 4 yrs Findings: Ht 150 cm

maintain the circulation it was necessary that the venous pressure exceed the intracardial pressure by at least 35 to 40 mm of water. The pulsations in the presence of a pneumopericardium, or of a bilateral pneumothorax, are identical with those observed when the chest is opened and the heart exposed.

(5 ft), wt 55 kg (121 lb) Congestive failure was present. The lt ventricle was much enlarged, the rhythm was regular, BP 180/110. Moderate degree of anemia, blood chemistry normal, Wassermann negative. Fundi: Copper wire appearance of retinal arterioles. ECG: T of lowered voltage in all leads.

*Roentgenogram, ant. view (A)* Large silhouette of aortic configuration. Slight degree of pulmonary congestion.

*Course* The patient improved temporarily on digitalis and diuretics. Cardiac failure reappeared temporarily. Low grade fever developed. Findings: The area of cardiac dullness was much enlarged, including the upper sternal area, the apical thrust was not palpated. Both a pericardial friction rub and gallop rhythm were heard. BP 160/98. Sedimentation rate 24 mm/one hr. Hb 10.5 gms, rbc 3.7 mill, wbc 7650 with 76% polys (nf 55, f 21). ECG: Sx, rate 116/min, no axis dev, digitalis effect (D).

*Roentgenogram, ant. view (B)* Size of the silhouette further enlarged, vascular pedicle shortened. Absence of pulmonary congestion.

*Course* Dorsal paracentesis of pericardial cavity was done, and a guinea pig inoculation was positive for tuberculosis. Low grade fever persisted, cough and paroxysmal dyspnea were present. Findings: The patient was weak, febrile and coughed. Ascites was present but other evidence of congestive failure was mild. A friction rub was not heard, the sounds were of low intensity. BP 146/90, Wbc 5800 with 89% Polys (nf 83, f 6). ECG: Sx, rate 114/min, right axis dev, digitalis effect (D).

*Roentgenogram, ant. view (C)* Miliary tuberculosis of lungs. Silhouette large, of aortic configuration.

*Course:* Death 3 days later.

*Postmortem:* The pericardium was thickened and tightly adherent to the epicardial surface of the heart in some areas, loosely adherent in others and entirely free in still other areas. 50 cm<sup>3</sup> of fluid were present. Thick caseous material covered the visceral pericardial and epicardial surface (E). The heart was much enlarged, the right ventricle dilated, the thickness of the right ventricle was 0.9 cm and of the left ventricle 1.8 cm (F). The endocardium of the left atrium showed several tubercles (arrows). The valves, coronary ostia and vessels were normal. Microscopic: Tuberculous pericarditis, toxic myocarditis. The pleural spaces were obliterated. The lungs were studded with miliary tubercles (G). Other organs showed sparse miliary tubercles but the peritoneum and G-I tract were free of them. Kidneys, microscopic: Early arterio- and arteriolar nephrosclerosis.



*Comment:* The patient had hypertensive cardiovascular disease and congestive failure. The roentgenologic silhouette was large and of aortic configuration. Tuberculosis of the pericardium with effusion developed that caused the silhouette to increase further in size and the vascular pedicle to shorten. The effusion gradually resorbed, the silhouette receded somewhat in size. Miliary tuberculosis developed and chiefly affected the lungs. This caused strain on the right ventricle, as evidenced by the appearance of right axis deviation in the ECG.

The direct observation of the beating heart in experimental animal work is misleading. This may be easily proved by comparing the fluoroscopic findings in a dog with closed thorax with those obtained by direct inspection with thorax open. They differ greatly.

If fluid is present it is seen to splash under the influence of the vigorous heart action, although the distended pericardial shadow itself shows no pulsatory transmission. As long as the cavity is entirely free, fluid collects in the deepest portions. Fluid-filled pockets, however, can be observed in case of partial adhesions or obliterations. Such pockets have been clearly demonstrated by means of lipiodol injections.

Pneumopericardium must be differentiated from collection of air in the mediastinum and from herniation of the lung. Neither mediastinal emphysema nor pneumothorax has the characteristic lateral delineation with origin at the level of the vessels. In the former case, air is seen to extend in the form of transparent bubbles into the soft tissue shadows of the neck. In the latter instance, air is found as a transparent area somewhere between the lung and the chest-wall. Herniation of one lung toward the opposite side, occasioned by a pneumothorax, displays an arc-like deviation of the mediastinal pleura, and under the fluoroscope one witnesses the respiratory to-and-fro movement of the arc (*in respect to the midline*). The increased tempo and dynamics of the pulsatory activity of the heart that are seen in the presence of pneumopericardium are also observed in the presence of a pneumothorax, provided air reaches the mediastinal surface. Since pneumothorax is usually unilateral, the change in activity is consequently limited to one side only. In none of the aforementioned conditions will it be possible, in contrast to pneumopericardium, to visualize adequately the lower heart border by changing the position of the patient.

**Tumors and Diverticula.** A localized, sharply defined, oval or hemispherical out-pouching somewhere along the contour of the silhouette may be an expression of varying pathology originating in the heart, pericardium or mediastinum. Formations of unicentric growth which expand within a pre-formed capsule, bulge in a globular fashion. This is the character of cysts, diverticula, hemangiomas of the pericardium, or of mediastinal growths (ganglioneuromata, dermoids) which are broadly attached to the pericardium. Malignant tumors of the pericardium, primary or metastatic, often reveal multicentric growth, and, as a rule, soon lead to effusion. In other instances they form a solid thick layer around the heart proper. The result is an enlargement of the silhouette and diminution or absence of pulsations. Actual expansile pulsations are observed in the rare hemangiomas. In addition, aneurysms of the aorta of intrapericardial origin, and cardiac aneurysms may be mentioned. The use of a diagnostic pneumopericardium is an aid to the differentiation between solid tumor formations located either inside or outside of the pericardial cavity and certain types of solid extrapericardial tumors and diverticula. This diagnostic procedure may be likened to the aid afforded by pneumothorax in differentiating between intra- and extrapulmonary location. True diverticula may reach the size of a man's fist. It is interesting that they are found in combination with cardiac enlargement and hydroperi-

cardium—perhaps enlarging secondarily to an increased intrapericardial pressure. Calcification was observed in cystic formations following traumatic hemorrhage. Diverticular formations due to encapsulated effusions will be discussed under constrictive-adhesive pericardial disease.

**Chronic Constrictive—Adhesive Pericardial and Mediastino-Pericardial Diseases.** **ANATOMY.** Local loose intrapericardial adhesions and even complete adhesions between the visceral and parietal layer of the pericardium, provided there is no thickening or shrinking or external fixation, interfere but little with the cardiac function, and characteristic roentgenological signs are not known. Other pathological forms are of great importance because of the resulting interference with cardiovascular function. They are not often diagnosed, and from the therapeutic point of view should be submitted to surgery. Clinically the existence of this pathology is not always considered. Symptoms and signs may be attributed either to associated cardiovascular disease or erroneously interpreted as sequellae of cardiac (myocardial) disease, or may be considered indicative of other than cardiovascular disease such as cirrhosis of the liver.

The subjective symptoms and the peripheral clinical findings of inflow stasis are usually more pronounced than the local findings over the heart region and the chest. Certain clinical signs such as systolic depressions of the chest wall are thought to be characteristic but are by no means unequivocal. The two main pathological forms which will be discussed for practical purposes are as follows: 1) the pericardium is represented by a solid, thick layer. The pericardial cavity is obliterated in part or completely, and the heart and large veins are encased, wholly or in part, and are under pressure because of the contracted state of the surrounding layers. 2) the pericardial cavity is obliterated partially or totally. The pericardial layer is thickened to varying degrees. External, firm, fibrous tissues and adhesions anchor the heart plus the adherent pericardium to the environment, i.e., the chest wall, spine, diaphragm, mediastinal, costal and viscera pleura. With the first form the heart function is handicapped mainly because the systolic-diastolic change in form and position is inhibited; the heart has been compared with a hand within a glove which is too small. The muscle itself is usually the seat of degenerative processes also. The heart remains either normal in size or is found to be abnormally small. With the second form the heart function is interfered with because resistance is offered to systole, and if systole does displace adjacent structures an elastic pull is exerted during diastole, especially by the elastic chest wall. Cardiac hypertrophy and dilatation may be associated. Both conditions display many variations and transitions and may be complicated by other cardiovascular disease.

**ROENTGENOLOGICAL FINDINGS.** The roentgenological description gives a summation picture but it should be remembered that only some of the signs are to be found in a given case. Static signs will be discussed first, dynamic signs later.

**STATIC ASPECTS** *Shape and Size.* The shape of the heart may be dominated by an underlying and often primary cardiovascular disease. The cardio-pericardial shadow is usually globular or pear-shaped and is in a median



position, unless pleuro-pulmonary pathology leads to displacement. There may or may not be present enlargement of the silhouette. Enlargement can be caused by: 1) an actual increase in the heart size; 2) by thick pericardial layers which may increase the measurement as much as 2 cm.; 3) by the presence of an old encapsulated pericardial effusion; 4) by mediastinal effusion. Any of these factors may occur in combination. Absence of enlargement, especially to the right, is quite impressive and striking for cases with the clinical picture of a typical right sided failure.

The constrictive effect has been clearly demonstrated in two instances where in the course of one and five years the L figures for the cardio-pericardial

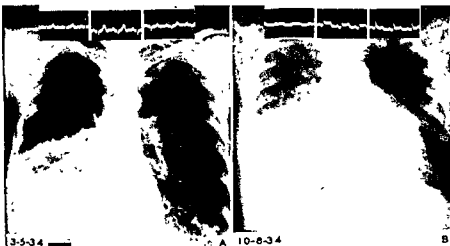


FIGS 285, 286—30 yrs, M. Pneumonia, empyema and purulent pericarditis at age 17, rib-resection anteriorly and posteriorly. No complaints. Normal cardiovascular findings. Cardiac pulsations seen and felt corresponding to area of anterior resection. *Ant view*: cardiac shadow slightly displaced to lt. Adhesions at lt. lung base. Contours of silhouette smooth. Normal appearance of pulsations. *Rt lat view*: ventral cardiac contour fuses with inner chest-wall contour in angular fashion, tentorial pericardial adhesion (wh arrow). Pleuro-pericardial adhesion. Clinical and roentgenological findings were unchanged over period of four years' observation.

shadow diminished as much as 2 and 2.2 cm. respectively. This constriction is associated with an enlargement of the left atrium in the presence of mitral stenosis. In the presence of aortic regurgitation, in such instances the characteristic roentgenological silhouettes.

The junctions of the silhouette are often less distinctly outlined than usual. A filling-out of the waist of the silhouette at the left leads to "mitralization." The vascular shadow may be foreshortened with a widening to the right occasioned by an enlargement of the superior vena cava, or by mediastinal effusion or thickening, and the aortic knob may be poorly visualized. The encircling process tends to affect the apical portion less or to even leave it free, and it then is observed to bulge like a hernia and simulate a cardiac aneurysm. One or both cardio-diaphragmatic angles can be bridged by broad, lateral, sharply defined adhesions. In the presence of such a bilateral process, the cardio-pericardial silhouette assumes a definite triangular shape in the anterior view.

Oblique and lateral views permit differentiation of such adhesions from costo-mediastinal adhesions, which are located in a plane definitely behind the heart. A partially or completely collapsed lower lobe with atelectasis or carnification is difficult to differentiate. Care must be used not to diagnose as an adhesive process the rather transparent and small triangular shadow which normally extends from the lower left border to the diaphragmatic contour. Since pericardial and mediastino-pericardial disease may be the local manifestation of



FIGS 287, 288—49 yrs, M. Cough for 10 yrs, becoming productive with blood streaks in mucus for last 3 months. Flatness at rt base. Clubbing. Cardiac findings negative. ECG essentially normal. *Ant view* (A) abscess cavity with fluid level in rt lower lung field, surrounded by large amount of abnormal density. Verification by Lipiodol instillation. Rt. lower cardiac contour in parts not visible, silhouette otherwise normal. Course operation 4 months later, rt posterior lobe found destroyed. Development of fever, tachycardia, orthopnea, rapid liver engorgement and marked increase in cardiac dullness. ECG: R-T segment shows high take-off in all three leads. *Ant view* (B) marked enlargement of silhouette, including vascular shadow. Rounded, prominent bulging at lt contour. Post-mortem: suppurative pericardial effusion with marked dilatation of pericardial cavity. Parietal pericardium connected by synechia to epicardium above apical portion of heart, cranial to it is large collection of pus, causing a marked bulging of lt upper portion of pericardial sac. Heart not enlarged, coronary arteries patent, parenchymatous degeneration of myocardium. Huge abscess cavity in rt posterior lobe with microscopical findings of carcinoma. Aspect of pericardial effusion with sacculations of exudate.

a polyserositis, one should search for roentgenologic signs of pleural disease, and include a search for interlobar affections by using the oblique views and placing the patient in a lordotic position. Tent-like shadow formations along the silhouette indicate a pathological process in the adjacent lung tissue or in the mediastinal or costal pleura. The pericardium is locally pulled off due to overlying tension, but generally the pericardial cavity does not reveal local alterations. Tent-like shadow formations along the diaphragmatic contour may express pleural adhesions but more often they represent localized elevations due to structural changes of the overlying broncho-pulmonary area.

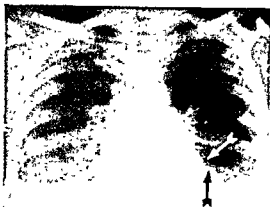


FIG 289—46 yrs, M Rheumatic fever at age 17 Since age 42 effort limitation, dyspnea, abdomen increasing in size Friction rub at age 45; since then leg edema Venous congestion with slight degree cyanosis and liver enlargement Edema, ascites, pleural effusion Cardiac dullness (after removal of pleural fluid) not appreciably enlarged; apical thrust not felt Absence of chest wall pulsation Heart sounds clear, embryonic rhythm, tachycardia, BP 95-110/80-85 Pulsus paradoxus ECG nsr, very low voltage all four leads Chronic constrictive pericardial disease Bilateral effusion partly removed, a small amount of air entered lt pleural cavity Ant view fluoroscopy reveals complete absence of any pulsations Contours of silhouette rather straight either side Rt. cranial vascular shadow (sup vena cava) prominent Absence of congestive lung failure. A mediastinal hydropneumothorax established; mediastinal pleura (wh arrow) and horizontal fluid level (bl arrow) are visualized Course operation (Dr. C S Beck, Cleveland): complete obliteration of pericardial cavity, scar measuring in some places 0.5 cm Adhesions between pericardium and sternum and between pericardium and rt diaphragm Pericardectomy Marked improvement. Were it not for the air collection, the shadow cast by the mediastinal effusion might have been interpreted as the cardiopericardial shadow proper Since cardiac

If air is introduced into the pleural cavity, these conic or pointed shadows may either disappear or may be intensified or may occasionally thus make their first appearance. Haziness of mediastinal contours is due to adjacent interlobar and pulmonary pathology. A marked obliteration or disappearance of the retrosternal or retrocardial area, or of both, is usually caused by mediastino-pericardial pathology, unless marked cardiac enlargement or lung disease accounts for it.

*Localized Encapsulation.* Exudate, rather inspissated in character, may persist and is usually found encapsulated. The presence of a larger and localized amount leads to localized bulging of a shadow mass, usually round and smoothly outlined, rarely polygonal in shape. The site of preference is on the right. First size is often attained, and faint transmitted pulsations may be present. The base, as seen in all views, is in broad contact with the cardiopericardio-vascular silhouette and its contours are continuous with it. It may well imitate an aneurysm of the proximal portion of the aorta and is identical in appearance with a true diverticulum of the pericardium. The diagnosis of such a sacculated exudate depends mainly on the general roentgenological diagnosis of chronic pericardial disease. Aneurysm is unlikely to be present if other visible portions of the aorta show neither widening or displacement.

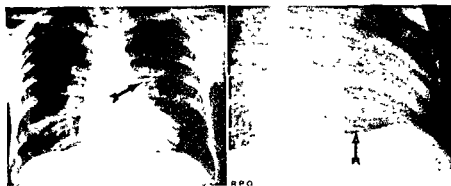
cance

An autobiographical description of this case is given by the patient, an M.D., in Penna. M J. 1936, 40, 18.

A localized obliteration of the pericardial cavity occasions peculiar changes of the silhouette in the presence of a pericardial effusion. Thus the circular

smooth outline of the left or right border may be replaced by out-pouchings with a depressed and flattened connecting contour between them.

*Postoperative Changes.* Surgery in chronic pericardial or pericardio-mediastinal disease consists chiefly in decortication of the thickened pericardial layers. Removal of parts of the anterior chest wall is indicated in some



FIGS 290, 291—19 yrs, M. Cardiac pain and palpitation on effort since age 15. Operation for Tbc, abscess on foot age 18. Ascites recently Slight cyanosis, liver engorgement, no edema Apical thrust not palpable, diastolic propulsion of precordium Whole lt. ant. chest wall shows systolic retraction and diastolic outward movement to lt Intensive dullness over lower sternal area Second sounds reduplicated, second part louder and simultaneous with diastolic propulsion BP 100/65. Rate 80 ECG n.s.r, no change of electrical axis in different positions Liver had presystolic and systolic pulse and diastolic collapse. Wassermann negative Adhesive mediastino-pericardial disease. *Ant view:* moderate enlargement of silhouette, L 14.1 cm, B 10.4 cm, T 14.2 cm, Th 25.2 cm Widening of rt. cranial vascular shadow (sup vena cava) Triangular shadow extending from lt cranial contour into interlobium (bl arrow) Moderate degree of congestive lung failure Fluoroscopy: amplitude of cardiac pulsations definitely increased, 0.6 cm at lt. border Lt. lower contour lifts with each systole Hardly any movement visible at aortic knob *Rt post obl view:* pulsating lime salt deposit at lower cardiac border (bl arrow). Typical clinical picture with predominance of the external adhesive type Calcification in pericardium Pleuro-pericardial adhesion The discrepancy between the increased cardiac and the decreased aortic pulsations is of diagnostic importance The stroke volume is obviously diminished The increased cardiac silhouette pulsations should be explained in two ways: (a) fixation of the atrio-ventricular septum and compensatory movement of the outer muscular wall of the heart, (b) the diastolic rebound of the elastic chest wall distends the heart beyond its diastolic position Fig 164 from Roesler, H, in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System, ed by Ross Golden, 1936, p 249 Courtesy Th Nelson & Sons, New York

instances. The following changes have been observed postoperatively: 1) a diminution of the previously enlarged pericardio-cardial silhouette; here thick layers had been removed or associated cardiac dilatation had receded 2) a diminution in the size of the vascular (superior vena cava) silhouette, and

were noted because only parts of the thick layers had been removed. 5) bulging or herniation of the left lower silhouette area; here only a part of the left ventricle was freed 6) disappearance of congestive lung failure due to an im-

provement of the hemodynamic conditions in the lesser circulation subsequent to freeing of the heart.

**Lung Fields.** Pleural effusions interfere with diagnosis, and removal by paracentesis should precede a second roentgenological study. The observation of the lung fields may reveal absence or but a slight degree of congestive lung failure. This fact is rather impressive in view of the more or less marked signs of congestive failure in the greater circulation. As an explanation it may be suggested that the disease process in question usually embarrasses the right side of the heart more than the left. This is demonstrated in many postmortem specimens, but even with the pathology equally distributed



FIG 292.—51 yrs, M Rheumatic fever and pericarditis at age 13 and 17. Valvular lesion diagnosed at age 21. Always hard working. Attacks of failure since age 38. At age 42 diagnosed as and operated for adhesive pericardial disease (rib resection); much improved. Congestive failure since age 50. Systolic retraction of lt. chest wall, diastolic propulsion of apical area. Cardiac enlargement. Mitral valvular lesion and aortic stenosis. BP 110/70. ECG: auricular fibrillation. Valvular lesion and adhesive mediastino-pericardial disease of rheumatic etiology. Ant view (illustration omitted): large silhouette with combined mitral and aortic configuration. Ring-like calcification along coronary sulcus. Fluoroscopy: pulsatory amplitude at lower contour increased, 0.3 cm, very small at aortic knob, absent at rt cardiac border and at coronary sulcus. Rt ant. obl view (illustration omitted): lt. atrium enlarged. Lt lat. view: lime salt deposits in annular arrangement, corresponding to coronary sulcus (wh arrows). Further deposits in sternodiaphragmatic angle.

Fluoroscopy: region of coronary sulcus not pulsating at all. Calcification of diaphragmatic pleura (bl arrow). Interlobar thickening (short bl arrows). The cardiac enlargement is caused by rheumatic heart and mediastino-pericardial disease. The increased pulsations along the lt cardiac border are not an expression of an increased stroke volume; fixation against the chest wall and fixation of the atrioventricular septum account for them. Deposit of lime salt occurred along the coronary sulcus only.

between the right and left side, the right ventricle will be embarrassed earlier. Inflow stasis back of the right heart likewise counteracts overfilling of the lungs. In other cases, however, congestive lung failure is present, and three conditions chiefly account for it. 1) left ventricular failure due to associated hypertensive or coronary artery disease; 2) mitral stenosis due either to intrinsic valvular deformation, or to an encircling of the base of the heart by constrictive and often calcified pericardial scar tissue; 3) embarrassment of the pulmonary vein circulation, possibly in the following indirect fashion. When the base of the heart is attached to the dorsal thoracic wall and when the lungs move freely with each inspiration, a strain will be thrown upon the



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**FIG 293**—67 yrs, F History Painless jaundice and rapid weight loss Findings Jaundice and enlargement of the liver were noted There was no evidence of failure and the heart was not appreciably enlarged Rate 88/min BP 138/80 Venous pressure 4 cm of water Circulation time, arm to tongue (saccharin) 18 sec, arm to lung (ether) 10 sec Laboratory evidence for biliary obstruction and liver damage E C G N.s.r, P deflections small, II axis dev, S-T segment depressed in I, II, T in CF. of low voltage, (no digitals)

*Li ant obl view* A large plaque-like zone of calcification is noted over the rt side of the heart

*Course* The operation revealed carcinoma of the bile ducts with metastasis to the liver

*Postmortem* (2 mos following the roentgenogram) A carcinoma of gall bladder wall and a subhepatic abscess were found Both pleural cavities were entirely obliterated by old fibrous adhesions There was a complete obliteration of the pericardial cavity by rather firm adhesions There was noted a lozenge-shaped calcareous plaque curving about the rt. atrium, measuring 7.5 cm in length, 4.5 cm in width, 1.5 cm in thickness, the inner surface was covered by the pectinate muscles of the atrium, which it infiltrated The weight of the heart was 400 gms The myocardium, endocardium, valves, coronary arteries were normal, a moderate degree of atherosclerosis of the aorta was noted Microscopic. Mild degeneration of the myocardium

*Comment* The localized calcification of the pericardium and of the adjacent portions of the myocardium was accidentally discovered on routine radiologic examination of the chest

cardiac root structures since the heart cannot follow the caudal and ventral propulsion of the lungs. Thus, during inspiration, the flow of blood in the veins may be retarded or stopped completely. Haziness of the lung fields is observed with pleural edema or thickening, and with pleural effusion.



FIGS 294, 295—46 yrs, M Shot in lt chest at age 25, thoracocentesis revealed blood Rheumatic fever at age 29 Hard working until age 44 Dyspneic for two years, ankle edema for one year Congestive failure in greater circulation Respiratory excursion of lt chest much diminished, intensive dullness posteriorly and laterally Heart action neither visible nor palpable No murmurs BP 120/70 ECG nst, T<sub>1</sub> isoelectric, T<sub>2</sub> diphasic Cardiac constriction *Ant. view* (recumbent position, Potter-Bucky diaphragm technique): extensive lime salt deposits corresponding to lt posterior lobe area with bullet in center. Cardiac shadow gobular, displaced to rt Ringlike calcification of pericardium (short bl. arrow) *Lt. ant. obl. view* (standard technique, with course of rays from rt and above to lt and downward) borders of lt. posterior lobe outlined by lime salt deposits, note interlobar fissure (wh arrow) Calcification of pericardium (short bl arrows). Fluoroscopy. cardiac shadow neither pulsates nor moves with respiration Bullet freely movable The etiology of the process is most likely traumatic in origin Hemothorax and hemopericardium lead to intensive calcification The bullet shifts within an encapsulated, old, mediastinal exudate A good demonstration of the anatomy of the lt posterior lobe.

**Calcification.** The deposition of lime salts is the most unequivocal sign of the presence of chronic pericardial disease but unfortunately, from the viewpoint of diagnosis, extensive deposits are not very common. The visible amount may vary from a few granules to an almost complete casing with a thickness as great as 2 cm Locations of preference are the coronary sulcus and both the diaphragmatic and sternal aspect of the right ventricular area. Then follows in order of frequency, the left ventricular area, its apical portion being often exempt; the area over the left atrium is rarely involved The deposits appear in the form of rings, plates, buckles, streaks and granules, radiating, arc-like, fork-like or linear; or branching and intercommunicating

... and smooth or irregular in outline. They are best observed and  
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calcifying process almost entirely involves the thickened pericardial layer, tangentially situated plaques will present themselves as encircling band-shadows along the silhouette. Where only localized plaques are present, their

peripheral site, in contradistinction to deep seated calcification, must be proven by adequate views in fluoroscopy and on roentgenograms. Simultaneous calcification of the cardiac and diaphragmatic muscle is not uncommon. It is very difficult, if not impossible to diagnose calcification in the coronary arteries in the presence of extensive pericardial calcification.

Calcified thrombi are represented by large, round or oval masses. Calcification limited to the apical portion, in conjunction with the history and clinical findings of coronary artery disease, must make one consider the presence of a cardiac aneurysm. Calcareous deposits in the fibrous skeleton of the heart and in the heart valves themselves are located centrally in the silhouette in all the views, and are characterized by their extensive movements. Calcified areas in the rib cartilages, within lymph nodes and bronchial walls, are found to project outside of the heart in certain views or may be located in stereoscopic roentgenograms.

Pericardial calcification does not necessarily indicate severe embarrassment of the cardiovascular function. It may have developed at a time before the obliterative and shrinking process occurred, and even may have prevented this malignant course. Very good function within this shell is then guaranteed. Or it may take place in a late stage, after obliteration and constriction are established. Then incrustation is only a special form of this condition though it may add to the embarrassment.

**DYNAMIC ASPECTS** *Types of Pulsations* Abnormal findings regarding pulsations are common. A decrease or even complete absence of the amplitude occurs commonly, but an increase is occasionally noted. Both types are well explained by the underlying pathology.

With constriction as the predominant feature, the extent of the amplitude diminishes or becomes nil. This may be observed all along the silhouette, or, while the greater part seems to be at rest, the apical portion may have preserved its normal activity. If pulsations are present, they lack the normal speed and wave-like character. The cardiac output per beat in such a case with constriction was found to be 18-26 cm<sup>3</sup>, as compared with the normal figures of 55-80 cm<sup>3</sup>. (average 65 cm<sup>3</sup>), and the cardiac output per minute was 36% lower than the average. Under the effect of exercise the heart rate in this patient increased from 106 to 144, while the output per beat remained almost exactly at the same level. One correspondingly finds the pulsatory amplitude, as observed during fluoroscopy, not appreciably altered. Diminished or absent pulsations may be found, however, without much circulatory embarrassment and without much diminution of the pulsatory output. Then a thick layer or shell is present, but the pericardial cavity itself is not obliterated or only obliterated in circumscribed areas. Calcareous deposits may or may not change their mutual relationship. In the latter case the armour is not the plate variety, so to speak, but rather of the chain or link type.

Such hearts with diminished or absent pulsations reveal a remarkable change during the stage of a total constriction. A case of this type is

presence of pneumopericardium and pneumothorax); but roentgenologic



follow-up studies show the persistence of these improved pulsations and they may even assume a normal aspect. This has been observed fluoroscopically in a number of cases and has been objectively demonstrated by means of kymograms. The work of the heart improves correspondingly as exemplified by the pre- and post-operative figures for such a case, with stroke volumes 36.9 and 88.8 cm<sup>3</sup>, and minute volumes 3.76 and 7.90 liters, respectively.

The fact that the circulation is maintained in spite of complete lack of pulsations along the cardiac silhouette proves the persistence of movements of the atrioventricular septum.

Very small pulsations are likewise associated with the heart in myxedema and with acute coronary occlusion.

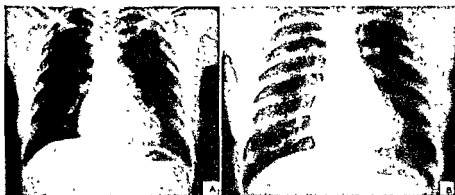
In other instances a great amplitude is observed, especially with a rapid diastolic outward movement. It reminds one, at first glance, of that seen in cases with a greatly increased stroke volume as in marked bradycardia. But the small amplitude along the great arterial vessels is in vivid contrast to the large excursions of the ventricular border, where the increased amplitude does not express an increased stroke volume but merely corresponds to an altered mode of ventricular contraction. It is found, first, when there are external adhesions, fixing heart and pericardium to the anterior chest wall in the presence of a well functioning heart muscle; second, when the base of the heart is fixed in the course of obliteration of the pericardial cavity. Here the longitudinal contraction of the ventricular cone is interfered with and this reduction in movement is compensated for by increased marginal excursions. Marked chest wall movements are noted clinically in these instances. In the first type ventricular systole deforms the left chest wall whose elastic rebound during diastole occasions the heart to expand passively. The following explanation can be offered for the second type. The aspiratory action of ventricular systole on the chest wall is not compensated for by the opposing centrifugal forces in the change of the shape of the heart, because the normal lever movement of the heart, the erection of the left lower pole region, is inhibited. The increased marginal excursions likewise create a marked aspiratory action on the chest wall. Since the systolic change in heart shape is interfered with, the chest wall does not receive a forward thrust during systole; consequently in the subsequent diastolic relaxation an opposite centripetal movement is not called forth. Hence, the centrifugal impulse due to diastolic filling of the ventricles can act unrestrictedly on the chest wall, and a diastolic outward thrust results. It should be understood therefore that the presence of external adhesions need not be postulated when these abnormal roentgenological and clinical pulsations are observed.

*Influence of Respiration and Body Position* Little diagnostic aid is obtained from the study of the mediastinal shadow in the lateral prone positions unless the respiratory influence is tested in addition. Changes in position of the mediastinal shadow may be observed in spite of an adhesive process, though we may actually deal with rotation only. A lack of shift of the left lower pole region, which normally would amount to 2 to 2.5 cm, to be evaluated only in the absence of any considerable cardiac enlargement.

As compared with a landmark outside the body, e.g., with the tip of a finger held against the fluoroscopic screen, the mediastinal shadow is observed

in many normal instances to rise toward the end of a deep inspiration. The rise of the mediastinal shadow therefore cannot be considered as a pathognomonic sign. One may observe, however, that one heart border rises while the other one is lowered, or that one rises more than the other. Further, one may notice that several denser band or streak-like shadow formations appear between the pericardio-cardial shadow and the diaphragmatic contour. Both of these findings indicate external adhesions.

The left lower pole region normally shows an inspiratory caudal and medial



FIGS 296, 297—30 yrs, F Stab wound of heart at age 25, followed by hemopericardium. Suture of heart wound, complete recovery. Clinical and electrocardiographic examination reveal no abnormalities. Ant view heart normal in size, shape and activity. (A) Taken during respiratory pause. Landmark corresponds to tip of finger resting on fluoroscopic screen. (B) Taken during deep inspiration. It is noted that silhouette has risen exclusively on its side. Landmark again corresponds to tip of finger on fluoroscopic screen. Lat view (illustration omitted) respiration does not bring about a change in size of retrosternal field. Post-operative adhesions between heart, pericardium and internal chest wall, most probable. While a slight to moderate rise of silhouette is physiological with thoracic type of respiration, such an asymmetrical rise is never observed under normal conditions.

the diaphragm while the lateral contours move and flatten out. In this connection spanning of the cardio-hepatic angles is commonly associated. Observation in the left anterior oblique view may reveal, for the whole cycle of respiration, a constant and therefore abnormal relationship of the dorsal silhouette border to the bi-diaphragmatic angle. All these findings lose much if not all of their significance in the presence of a large silhouette. Finally a marked straightening of the silhouette borders during forced inspiration has been observed in adhesive mediastinal disease.

When adhesions fix the pericardium to the lungs at each side, and to the chest wall in front and behind, they interfere with the normal ventral and cranial shift of the anterior chest wall. On inspiration the roots of the lungs are not moved, the chest expands laterally and is lifted together with the shoulders. The central portions of the diaphragm and its crura move but

little and the abdomen, instead of protruding, is drawn in. A lateral view reveals then, clinically or roentgenologically, an inspiratory cranial and ventral movement of the uppermost contour of the chest wall profile, while the middle and lower portions of the profile, approximately the caudal half of the sternum and the upper abdominal contour, move in an opposite direction, i.e., dorsad. Inspiratory and expiratory contours cross each other. This is the sign of the crossed profile. The retrosternal triangular transparent area should also be observed in this lateral view. Under normal conditions it will change in size and shape during the act of respiration. But since cardiac enlargement and obliteration of the medial pleural sinuses may also prevent this change, little diagnostic value can be ascribed to the absence of this phenomenon.

Valuable information may be gained by the study in the recumbent, lateral positions. The changes of the mediastinal shadow in position and shape, and its return toward the midline during inspiration have previously been described in chapter III. With fixation in front and behind, the mediastinum resists displacement by gravity, and therefore will not rise from the dependent side during inspiration. This study must be carried out for both the left and right lateral positions with the patient breathing deeply. An absence of a shift away from the dependent sides is suggestive of broad, external fixation. There are other conditions, however, that will reveal the same findings: bilateral pleural adhesions, a marked degree of pulmonary congestion associated with pleural effusions, much hydropericardium, much cardiac enlargement, and the inability of the patient to breathe deeply. Nor does the presence of the mediastinal shift permit the exclusion of localized external adhesions. A lack of change in shape of the silhouette, when carrying out the manoeuvre, is also suggestive of broad, external fixation; but a markedly hypertrophied and dilated heart fails to show such change.

Little use has been made of the Valsalva and Muller tests though their application holds some promise.

In conclusion, the evaluation of the roentgenological signs becomes increasingly difficult when the silhouette is considerably enlarged. It is also complicated by the presence of other basic cardiovascular disease. Presence of calcification is then the most reliable positive sign.

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## CHAPTER XII

### CONGENITAL CARDIOVASCULAR MALFORMATIONS

Both the roentgenologic and clinical diagnosis of congenital cardiovascular malformations are often difficult because certain defects may exist without roentgenologic indication of their presence and others, such as cardiac enlargement or prominence of the pulmonic arch, cause changes which are not characteristic for congenital malformations alone. Moreover, there are often combinations of different types of congenital cardiovascular defects, and occasionally acquired valvular lesions are superimposed. It is a mistaken idea that congenital cardiovascular disease is observed only or mainly in the

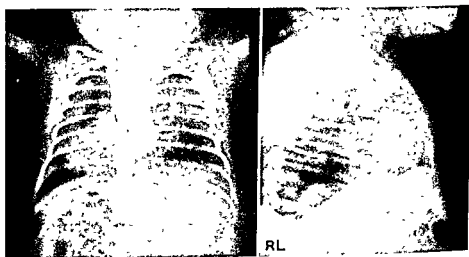


FIG. 298—1 month, M. Increasing cyanosis since birth. Transposition of all organs. ECG: characteristic inversion lead I, superimposed rt axis dev. *Ant. view*: cardiac silhouette directed to rt, of boot-shaped appearance. Supra-cardiac shadow slightly enlarged. Liver at lt., stomach at rt. *Rt. lat. view*: absence of thymic enlargement. Congenital cardiovascular malformation in the presence of transposition of all organs.

young and that cyanosis is the outstanding feature. Many cases are never  
 " and pass through life classified as rheumatic heart disease.  
 , the erosion

The roent-  
 genologic study of congenital malformations in infants renders comparatively  
 little diagnostic aid because the silhouette contour is not as yet differentiated.  
 Later in life there are fewer types of malformations to be considered, because  
 certain types of them lead to death at an early age, but then a more detailed  
 roentgenologic study is possible.

In a group of 68 cases with an average age of 16.8 years, there were present  
 positive, abnormal roentgenologic findings in 85% of the cases. These findings

were of definite diagnostic value in 51% of all the cases and in 60% of the cases with positive roentgen findings. Abnormalities in the pulmonic area were approximately three times more frequent than in the aortic area.

Further diagnostic progress may be expected from the recently introduced method of intracardiac visualization by means of contrast media.

**Dextrocardia.** This term simply implies that the greater portion of the heart lies in the right chest. This may be the result of a displacement dependent upon an extrinsic cause and acquired either during fetal life or, more commonly, in the course of the post-natal period. It is also designated as dextroversion. Generally speaking, the volume of the right chest space is diminished and the volume of the left increased. The roentgenological study demonstrates asymmetry of the chest, abnormal findings of the lungs, pleura and diaphragm and some displacement of the mediastinum. The axis of the cardiac mass has undergone a corresponding shift or rotation.

In another group of cases, the chest is symmetrical and the lungs, pleura and diaphragm are normal but the heart is situated in the right chest. Two main types represent dextrocardia in the narrower sense.

One type consists of transposition of all organs. The appearance of the cardiovascular shadow is reversed (a mirror image) as is the architecture. The anterior view corresponds to the posterior view of a normal chest roentgenogram, and right and left oblique views are respectively reversed. Congenital or acquired lesions may be superimposed and modify the roentgen ray findings.

The other type is the isolated dextrocardia. Here the other organs of the body show normal arrangement or there may be a partial transposition present. This dextrocardia is by no means an anatomical entity. Although the anatomic study always reveals the presence of cardiovascular malformations, yet it may be impossible during life to diagnose their presence by clinical or roentgenologic methods. Functional capacity and prognosis depend upon the type of malformation. A normal arrangement of the chambers may be present with any one of, or a combination of, the following defects: transposition of the large vessels, pulmonary artery stenosis or atresia, defects of the septa, and right sided aortic arch. If a partial inversion is present, one or several of the four embryological segments may be



FIG 209—11 yrs, F No complaints Abnormalities of fingers and toes. Pulsations in right intercostal spaces Normal heart sounds BP 85/70 Normal chest and abdominal findings ECG essentially normal Ant view cardiac shadow in rt. chest cavity with atypical configuration. Aortic knob in normal position, to lt Pulsations of ventricular type noted along rt contour Cervical rib on lt Absence of pleuro-pulmonary disease Rt leaf of diaphragm lower than lt Abdominal situs normal Congenital isolated dextrocardia

involved: bulbus, sinus, primary atrium, primary ventricle. There is never a complete, mirror-like inversion. The aforementioned malformations are concomitantly found. The aortic arch may have a left or right sided course.

This short description gives some idea of the variability of the clinical and roentgenological findings.

Concerning the course of the aorta, the following observations hold true: when the cardiac shadow is of an atypical shape, i.e., does not represent a mirror-like picture, the aorta crosses to the left, and this combination is relatively common. However, if the cardiac shadow represents a mirror image of the normal silhouette (and be it understood that this whole discussion refers to a normal situs of all the other organs), the aorta may cross either to the left or to the right, but more frequently it crosses to the right. A typical left lower pole (apical) region is present only when it is formed by the right sided ventricle. It is indefinite or missing when the left sided ventricle or an

atrium form the right lower border of the silhouette. The aortic arch in all known cases shows a normal relation to trachea and esophagus in contrast to persistence of the right sided aortic arch which is found in combination with a normal situs of the heart.

A subgroup of isolated dextrocardia shows congenital defects of ribs, pectoral muscles and herniation of the lung

The right leaf of the diaphragm is found to be farther caudad than the left, although in a few instances they may be at the same level. In other words, we learn from cases of isolated dextrocardia that it is the position of the heart which governs the difference in the height level of both leaves of the diaphragm and not the liver as is commonly believed. Another fact substantiates this statement; in instances of isolated transposition of the abdominal organs, where both liver and heart are found at the left, it is then the left leaf of the diaphragm which is noted to be more caudad.

#### Interventricular Septal Defect.

When the defect is very small, there is little or no change in the shape of the heart; some minor degree of hypertrophy and dilatation of both or, predominantly, of one ventricle is



FIG 300—6 yrs, M Known to have heart lesion since early infancy. Marked cyanosis. Both cardiac and liver dullness at left. Right testicle lower. Systolic murmur over entire heart. ECG. nsr, rt. axis dev. Ant view cardiac and liver shadows at

common. The large vessels may be normal or there may be a slight dilatation of the pulmonary artery. Very large defects lead to considerable

cardiac enlargement but are often complicated by transposition of the large vessels. When the defect of the interventricular septum is associated with dextroposition of the aortic orifice, hypertrophy and dilatation of the right ventricle, and dilatation of the pulmonary artery and its branches, this combination is called the Eisenmenger complex. It seems that enlargement of the right ventricle and pulmonary artery are functional sequelae to a sufficiently large shunt from left to right. The frequent finding of arteriosclerotic changes in the lesser circulation also points to an extra load. It is conceivable, however, that the enlargement of the pulmonary artery represents an associated anomaly, i.e., an abnormal division of the truncus arteriosus communis. When stenosis or regurgitation of the pulmonic valve or patency of the ductus arteriosus are noted, enlargement of the pulmonary artery should be related to these findings. Clinical symptoms are usually absent with small-sized defects, and full physical efficiency is preserved. And although the harsh mesocardial murmur, often accompanied by a thrill, is very impressive, the roentgenologic findings are either entirely negative, or there is noted a moderate enlargement, globular in type, which is sometimes associated with a slight prominence of the pulmonic arch. The hilar vessels are of normal appearance and the left atrium is not enlarged. The amplitude of the pulsations may be moderately increased, often not more than one might expect in younger persons with a vigorous heart action. A marked increase, however, is noted when heart block is present. A very marked prominence of the conus, pulmonic arch and, in some cases, also of the intrapulmonic vessels, together with a considerable right sided enlargement must make one consider the presence of an Eisenmenger complex or associated anomalies.

**Interatrial Septal Defect.** Cases with a patent foramen ovale or with very small defects do not disclose any roentgenological signs and are not considered in this discussion. A defect of considerable size leads to a hydraulic disturbance, a left to right shunt, as a result of which the right side of the heart and the lesser circulation carry an increased amount of blood while a decreased amount is carried by the left heart and the greater circulation. The hearts are large; often of enormous size. This is due to a right sided enlargement, a dilatation far exceeding hypertrophy, and the right heart forms the entire anterior wall. The pulmonary artery is always larger than the aorta, the average

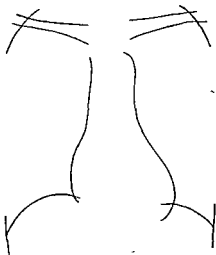


FIG 301—26 yrs, M No complaints Appeared for student athletic examination Heart not enlarged Loud, rough, blowing systolic murmur, maximum of intensity at crossing of left parasternal line and 4th intercostal space, a systolic thrill here Rate 82, B.P 100/85 ECG normal findings Typical interventricular septal defect Roentgenological findings normal as to shape and pulsations L 13, B 100, T 11.6, Th 23.2 cm.

ratio being 3:2. Expressed in absolute figures, the pulmonary artery is large or the branches of the e, and trunk and



FIG 302—14 yrs, F. Cardiac invalid since age 7. Underdevelopment, pallor, dyspnea. Precordial bulge, huge cardiac dullness, systolic retractions in intercostal spaces. Systolic venous pulse. E.C.G. auricular fibrillation, rt axis dev, slight. Ant view large pear-shaped silhouette L 188, T 187, Th 216 cm. Aortic knob not visible. Enlargement to rt and lt craniad. Intrapulmonary branches prominent, absence of congestion. Oblique views (illustrations omitted). Lt atrium enlarged, aortic arch not visible. Postmortem absence of pericardial adhesions. Rt atrium and rt. ventricle, especially conus arteriosus, much enlarged. Circumference of aorta 54 cm, of pulmonary artery 9 cm. Interatrial septal defect 1.3 x 4 cm. Mitral and tricuspid valvular lesion, slight degree. An example of great degree of right sided enlargement; dilatation due to a congenital intracardiac left to right shunt. From Roesler, H., *Arch Int Med*, 1934, 54, 339. Courtesy Am Med. Assoc. Press publishers.

branches are often the seat of atherosclerotic processes. The clinical diagnosis is complicated by the fact that in about 75% of the cases valvular lesions are present which predominantly affect the mitral orifice. This, however, does not modify the anatomical and roentgenological picture, it rather exaggerates it. The bearers of this condition usually reveal physical underdevelopment. Certain signs and symptoms caused by the presence of a large heart may mislead the clinician to diagnose adhesive pericardial disease.

The roentgen findings are quite characteristic. The cardiac shadow is large. This enlargement extends to each side but predominantly to the left so that the left lower contour may reach the left thoracic rib cage. The general configuration is globular, with a considerable cranio-caudad development and with a more or less marked prominence in the region of the pulmonary conus. Oblique views may or may not reveal enlargement of the left atrium. All of the branches of the pulmonary artery are enlarged but are sharply defined. Increased pulsations may be noted along the main branches in the hilus region. Normal transparency of the lung fields is present unless marked congestive failure takes place. In the majority of cases the aortic knob is either very small or entirely missing. In most instances, a low position of the diaphragm is noted.

It is not sufficiently realized that the enlarged right ventricle is situated predominantly or entirely to the left of the midline and thus causes either a predominant or almost exclusive enlargement of the silhouette to the left. When an enlargement of the left atrium is found it should be attributed first of all to a concomitant mitral valvular lesion. The enormous enlargement of the hilus vessels is often misinterpreted, tuberculosis and tumor being the chief er-

aneous diagnoses. In one instance the roentgen ray report seemed to support the clinical diagnosis of mediastinal tumor since the observed masses were sharply defined and did not pulsate. They were incised at operation and the patient bled to death. The postmortem examination revealed the presence of an interatrial septal defect together with an aneurysmal dilatation of the pulmonary artery and thrombosis of its branches.

An increased amplitude of the pulsations of these branches need not be an expression of valvular regurgitation but may also be produced by an increased pulse pressure in the lesser circulation with or without additional pathological changes in the wall. The low position of the diaphragm is, in part, a simple weight depression and in part a physiological adaptation to increase the available space for the lungs.



FIGS 303, 304—46 years, F. Palpitation since early childhood. Height 148 cm, weight 42 kg. Slight degree cyanosis, no clubbing, absence congestive failure. Marked cardiac enlargement to both sides and it cranial. Short systolic thrill in 2nd and 3rd intercostal spaces. P<sub>2</sub> much accentuated, followed by very short diastolic murmur. Peripheral vessels thin. B.P. 110/85, ECG n.s.r., rt axis dev, slight degree. *Ant view* marked enlargement of whole silhouette with characteristics of enlargement of rt chambers and marked prominence of region of pulmonary conus and artery (bl arrow). Aortic knob very small (hypoplasia) (wh arrow). Enormous enlargement of all intrapulmonary arterial branches as noted in longitudinal and cross sections. Rt ant obl view (illustration omitted): lt. atrium not enlarged, mass of heart mainly in front. *Lt ant obl view*: lime salt deposits noted along cranial contour of pulmonary artery (bl arrow). Congenital cardiovascular malformation, interatrial septal defect. Enlargement of rt side of heart and of vessels of lesser circulation. Calcification in wall of pulmonary artery.

The characteristic roentgen findings should be studied together because each of them, taken by itself may be found in some other pathological condition. The absence of the aortic knob, at the left side, is noted in the presence of an enormous enlargement of the pulmonary artery, in instances of marked mitral valvular lesions, with persistence of a right sided aortic arch, or in certain cases of coarctation of the aorta. Enlargement of the pulmonary artery and its branches occurs in association with certain other congenital anomalies such as patency of the ductus arteriosus and pulmonary stenosis; or it may arise from primary athero- and arteriosclerosis or syphilitic or mycotic



disease. The combination of pulmonary valve lesion and pulmonary artery enlargement, right sided cardiac enlargement, hypoplasia of the aorta and interventricular septal defect gives an identical roentgen appearance.

Various types of congenital malformations, those aforementioned as well as others, may of course complicate and modify the picture.

**Patency of the Ductus Arteriosus.** It occurs much more frequently in combination with other typical congenital cardiovascular malformations than without them. For didactic purposes, only the isolated form will be considered. The clinical and roentgenological diagnosis is more difficult in infants than in adolescents or adults. The importance of the correct diagnosis rests upon the facts that, in spite of good function, the high incidence of bacterial endocarditis entails a cautious prognosis and that surgical ligation should be considered when the diagnosis has been established.

The anatomical findings are not uniform; influencing factors are: the size of the ductus and its orifices, its direction, the age of the patient, the duration of the shunt, and the possible combination with dilatation of the pulmonary arterial system. With the latter condition, both acquired and concomitant congenital lesions occur. Other acquired cardiovascular lesions may modify

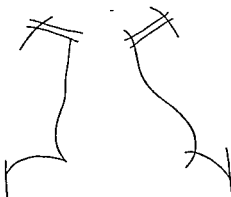


FIG. 305—31 yrs, F Dyspnea since childhood. Continuous systolic-diastolic murmur and diastolic thrill over pulmonic area B P. 120/85 Patient developed subacute bacterial endocarditis one year later. *Ant view* (recumbent position). moderate degree of enlargement. L 15, B 10 2, T 14 5, Th 23.7 cm. Nothing unusual as to pulsations Postmortem: enlargement of lt ventricle; subacute bacterial endocarditis of aor-

the roentgenologic and anatomical picture, affecting exclusively or predominantly either the right or left side, or both equally. When the intra-aortic pressure is well transmitted into the intrapulmonary system and when a sufficient amount of blood is short-circuited through the lesser circulation, both sides of the heart will develop some degree of hypertrophy and dilatation—the right side to overcome the additional pressure, the left side to manage the additional amount of pendular blood which is shunted through the lesser circulation, and possibly also from increased work; for otherwise the minute volume in the systemic circulation would be too small, blood being diverted from it (see above). Actually the heart may reveal a normal size, hypertrophy exclusively, or a varying degree of hypertrophy and dilatation which may affect both sides to a varying degree. Large hearts are rare, in such instances there is discovered either an

ray findings were of no diagnostic help

unusually wide communication between pulmonary artery and aorta or an extensive athero- and arteriosclerotic process in the lesser circulation, or a combination of both.

In size the aorta may be equal to or smaller than the pulmonary artery. In

rare instances the aorta may be small proximal to the ductus but larger distal to it. In a few instances a calcified plaque occurs at the point of attachment of the ductus. The anatomical findings of the pulmonary artery and of its branches are variable. The size of the artery may be normal, in spite of concomitant enlargement of the heart. Or it may be enlarged, from a moderate degree up to the size and shape of an aneurysm and the process may be limited to the portion proximal to the ductus or may involve any or all the branches. Combinations with regurgitation of the pulmonic orifice and with athero- and arteriosclerosis are observed. The ductus itself may be the site of an aneurysm.

It is obvious that an unequivocal, characteristic roentgenologic appearance cannot be expected for all cases. Most commonly, in the anterior view, one notes a slightly or moderately enlarged silhouette without prominence of the left atrial contour in oblique views. The waist of the silhouette has not completely disappeared. The pulmonic arch is moderately prominent and encroaches somewhat upon the aortic knob which otherwise is of normal size. The hilus vessels are either of normal appearance or, more frequently, slightly enlarged. The pulsations along the silhouette are rather vigorous, especially in the region of the aortic knob and the pulmonic arch. The left anterior oblique view reveals in some instances the window of the aorta overshadowed in parts. This finding, when other mediastinal disease and aortic aneurysm is excluded, indicates that the ductus is of

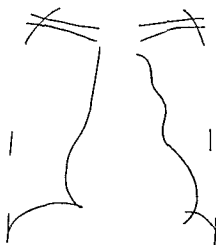


FIG 306—28 yrs, F. Dyspnea on effort and palpitation since age 23, increasing recently during pregnancy. No cyanosis. Slight ankle edema. Marked enlargement of heart. Short systolic apical and basal murmur  $P_2$  accentuated. No diastolic murmur. Rate 92, regular. BP 115/90. Ant view. marked cardiac enlargement, L 15.8, B 11.4, T 16.2, Th 23.5 cm. Marked prominence of pulmonic arch. Hilus vessels slightly enlarged. Lungs clear. Pulsations along the silhouette definitely increased. Rt ant obl view (illustration omitted). Lt atrium moderately enlarged, rt ventricle and conus arteriosus prominent ventrally. Cause death from peritonitis following Caesarian section. Postmortem persistent ductus arteriosus, 0.9 cm wide direct communication between aorta and pulmonary artery. Circumference of pulmonary artery 9.2 cm, of aorta 7.5 cm, above valves. Both ventricles much hypertrophied and slightly dilated. Both atria slightly hypertrophied and dilated. Mild degree of mitral valvular regurgitation. The roentgen ray findings in this instance were much more characteristic than the clinical findings.

considerable size. Rarely is there a marked prominence of the pulmonic arch present during childhood. It is uncommon to find a huge out-pouching of the pulmonic arch, the intrapulmonary branches may or may not be enlarged. The combination of enlargement of both trunk and branches permits one easily to ascribe the bulging at the cranial left contour to the pulmonary artery or to the conus or to both, in absence of branch dilatation, however, the differential diagnosis of a left-sided aneurysm of the ascending aorta may become difficult or

impossible. The perforation of an aneurysm of the ductus with secondary encapsulation is the occasion of the presence of a hazily outlined mass above the left hilus. Marked expansile pulsations of the contours of trunk and branches are observed only in the presence of complicating pulmonary valve regurgitation which may be either acquired or congenital. Diminution of pulsations, unless explained by severe heart failure, must make one think of endocarditic and thrombotic occlusion of the ductus. The roentgen ray evidence of localized calcification near the caudal aspect of the arch of the aorta, in young individuals, in whom the presence of calcification from ordinary atherosclerosis is unlikely, should be looked upon as a confirmatory sign, particularly if other clinical and roentgenological signs of patency of the ductus arteriosus are available.

A machinery murmur and thrill in the second and third left intercostal space are often, though not regularly present. No direct relationship exists between these clinical signs and the degree of pulmonary artery prominence. As a matter of fact, most pronounced clinical signs are compatible with negative roentgen ray findings (small opening of the duct), while on the other hand, in the absence of any clinical signs, marked visible dilatation may be found (large opening).

The roentgenological differential diagnosis must include the different types, as outlined above. Moderate prominence of the pulmonic arch, slight increase in the heart size and vigorous activity is likewise found in the presence of thyrotoxicosis. A prominent pulmonic arch is also found in the rare instance of stenosis of the pulmonic orifice with post-stenotic dilatation of the artery. The interatrial septal defect is in general characterized by a much larger heart and by the absence or smallness of the aortic knob and always discloses very large intrapulmonary vessels. The diffuse obliterative endarteritis of the lesser circulation has a similar appearance except that the aortic knob is of normal size. Patency of the ductus alone rarely if ever leads to enormous cardiac enlargement, and the presence of a very large trunk and branches occurs only in combination with and complication by aneurysmal formation.

Instructive studies have been recently carried out on animals with respect to the fetal circulation and the closure of the ductus arteriosus. 1. Sheep fetuses from 100 to 145 days in age (the full term is 147 days) were delivered by Caesarean section. The intra-uterine conditions were preserved as far as possible, leaving the umbilical cord attached to the placenta and covering the snout of the fetus with a rubber bag containing amniotic fluid in order to prevent respiration. The passage of radiopaque media was studied by means of direct and indirect roentgen cinematography. The whole of the superior caval blood is seen to pass into the right ventricle and through the pulmonary orifice into the pulmonary trunk, from this vessel it diverts in two directions. 1) into the pulmonary arteries and 2) via the ductus arteriosus into the descending aorta. Since the brachiocephalic artery leaves the aorta proximal to the entry of the ductus arteriosus, the superior caval blood does not pass to the coronary system, the head and fore-limbs. The inferior caval blood has a double course through the heart. The main part goes through the foramen ovale to the left atrium and ventricle and thence passes into the aorta and

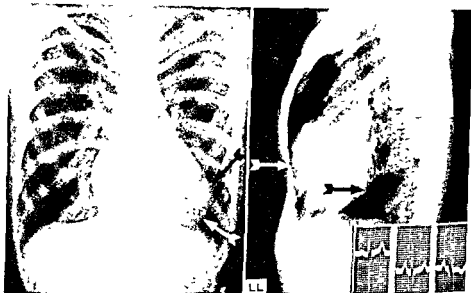
coronary and brachiocephalic arteries; hence the heart and brain are given preferential treatment with respect to the supply of oxygenated blood coming from the placenta. A minor part of the inferior caval blood passes with the superior caval blood into the right ventricle, thence into the pulmonary arteries and, via the ductus arteriosus, into the descending aorta. 2. Full-term lambs were delivered by Caesarean section 25 sec to 33 min after delivery, direct roentgen cinematograms were taken of the ductus arteriosus by using a radiopaque medium. It was demonstrated, that there were marked differences between the pictures obtained in vivo and those revealed by postmortem dissection; that the functional closure of the ductus had occurred in every case within a minute from delivery, and that even when the vessel was patent the flow through it appeared to be sluggish; that this functional closure was not effected by a rapid and simultaneous contraction of the total musculature of the ductus but through the independent and intermittent activity of groups of muscle fibers which led to twisting and kinking of the vessel; and that the functional closure of the ductus was apparently unrelated either to the discontinuation of the maternal blood supply or to the actual entry of air into the lungs of the lamb.

**Pulmonary Stenosis.** Only two important types of pulmonary stenosis will be considered here, one with a closed interventricular septum, the other with a defective one. The site, degree and extent of the stenosis are variable. In both stenosis of the conus arteriosus and of the pulmonary orifice the trunk of the pulmonary artery may be normal in size, hypoplastic, pencil or thread-like, or markedly dilated together with a thinning out of the vessel wall itself. The dilatation extends no farther than the bifurcation of the artery. With a proximal low position of the stenosis within the conus, its upper portion may be dilated and the lower portion contracted. With a low position of the stenosis at the pulmonary orifice, the upper portion of the trunk of the artery is dilated and the lower portion contracted.

is c

tion of the artery is not yet fully understood; it seems to be due to congenital hypoplasia of the wall. In animals, an experimental ligature around the base of the pulmonary artery leads to a poststenotic dilatation and thinning of the wall, and the microscopic examination reveals a marked reduction in the size of the media. The intrapulmonary branches are often rather small, although if a stenosed trunk is present they are relatively wide. A sufficient size of these vessels is guaranteed by the presence of an associated patent ductus arteriosus, or by the establishment of a collateral circulation which carries blood from the aorta or from its main branches into the lungs, through normal vessels such as the bronchial arteries or anomalous vessels. The heart itself is the seat of marked right ventricular hypertrophy, often with little dilatation, thus, the size is often nearly normal, sometimes moderately increased. The right ventricle takes part in the formation of the left lower pole region. Very little of the left ventricle is seen from the front. The left lower pole region reveals a peculiar shape. Instead of being conical, it is blunt, as though cut off. Two thirds or more of its lower contour is made up by the right ventricle and the remainder by the left ventricle. The pole is thus formed by the blending of the apical regions of both ventricles. The left

ventricle extends for a rather short distance along the cranial ventricular contour, mesiad from and craniad to the upper apex. When an interventricular septal defect is associated, this finding being much more frequently present than not, the aortic orifice is dextroposed so that the aorta rides over the defect. These findings, together with *right ventricular hypertrophy* and *pulmonary stenosis* represent the most common type, known as the tetralogy



FIGS. 307, 308—23 yrs, M. Cyanosis and clubbing since childhood. Was capable of athletic activity until age 20. Slight degree of dyspnea. Intensive cyanosis, clubbing. Lower portion of sternum flat on percussion, with systolic forward lift. No thrill. Blowing systolic murmurs at lower end of sternum and 3rd lt intercostal space. Rbc 12 mill, hb 150%. ECG: n.s.r, rt axis dev. *Ant view*: silhouette moderately enlarged with peculiar angulation at lt lower contour, so-called "coeur en sabot." *Cranial contour* (bl arrow) corresponds to lt ventricle which has been rotated craniad and dorsad by rt. ventricle. The latter forms part of lt silhouette above diaphragm (wh arrow). *Rt cardiac contour* elongated, of increased curvature. *Lt lat. view*: mass of heart bulges ventrally, corresponding to rt ventricle (wh arrow). *Lt ventricle, dorsally*, of normal appearance (bl arrow). Postmortem: tetralogy of Fallot: pulmonary stenosis, interventricular septal defect, rt ventricular hypertrophy, dextroposition of aortic orifice.

of Fallot. Here the heart is transversely situated and the region of the pulmonary artery and conus are rather depressed, even in the presence of a normal sized or slightly dilated pulmonary trunk. With the ventricular septum closed, the position of the heart is less transverse (more vertical) and the region of the conus is more or less prominent. With the tetralogy of Fallot the degree of torsion of the two great vessels is diminished, hence we are really dealing with one particular type of transposition of the great vessels. Why the waistline remains preserved with this anomaly while other types of heart disease showing *right ventricular enlargement* (mitral stenosis, cor pulmonale, isolated pulmonary stenosis, interatrial septal defect) show a fullness of the waistline, is explained by the peculiar hemodynamics in this condition. It will

be remembered that the axis of the normal right sided outflow tract approaches the perpendicular to the diaphragmatic level. With the tetralogy of Fallot the powerfully developed crista supraventricularis deflects the blood current so that it does not flow in the normal direction of the outflow tract, which

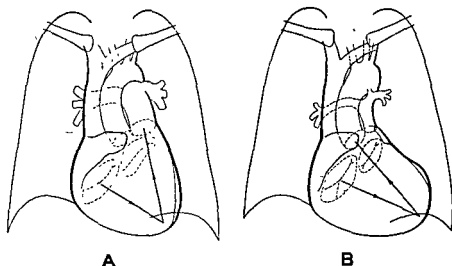


FIG. 309.—Schematic presentation of the roentgenologic anatomy in a case with mitral stenosis—A—and in a case with tetralogy of Fallot (pulmonary stenosis, right ventricular hypertrophy, dextroposition of aortic orifice with the aorta riding over an interventricular septal defect)—B—. The arrows indicate the course of the inflow and outflow tract for the right ventricle.

In—A—the direction of the outflow tract is nearly perpendicular to the left leaf of the diaphragm. Its lengthening and thickening favors the filling out of the heart's waistline by the conus and the pulmonary artery, the left lower border descends steeply. Thus the mitral configuration results.

In—B—the direction of the outflow tract is oblique from left-ventral-caudal to right-dorsal-cranial. The prominent supraventricular crista, i.e., the muscle layer between the pulmonary and aortic orifices, causes the blood current in the right ventricle to deviate toward the aortic ostium. The lengthening and thickening of the outflow tract results in a prominence of the heart to the left, the right ventricle forms the left lower pole while the left ventricle is displaced dorsad. The conus and pulmonary artery are narrow. The heart's waistline is preserved. Thus the *cœur en sabot* shape results.

Right ventricular hypertrophy is noted with both lesions but a difference in contours results because of the difference in the hemodynamic conditions. Courtesy Doz. Dr. E. Zdansky, Vienna and J. Springer, publisher, Vienna. From *Röntgendiagnostik des Herzens und der grossen Gefässe*, 1939, Fig. 185.

would favor the development of a prominent conus of the right ventricle, but flows instead toward the aortic orifice and thus the axis of this new outflow tract lies more obliquely, i.e., from ventral-caudal-left to dorsal-cranial-right. The development of this outflow tract, due to the altered hemodynamics, changes the shape of the heart: the waistline remains preserved, the apical portion formed by the right ventricle bulges and the left ventricle is displaced dorsad.

One learns from this discussion of anatomical findings, that a single characteristic roentgenological appearance is not to be expected

The most common finding is the unusual appearance of the left lower pole region; it appears plump, blunt, almost straight for some distance. It forms a deep and acute angle with the diaphragmatic contour. Cranially and medially the change into the rest of the contour is rather sudden and causes a marked angulation. As a result, the silhouette of the heart shadow, as it extends into the left lung field, reminds one somewhat of a sheep's nose. This configuration has been termed *cœur en sabot*, or wooden shoe heart. The cardiac shadow itself is often only slightly enlarged, predominantly to the left. The general configuration of the heart can be tentatively classified into two types.

One shows the waist of the heart markedly pronounced as a concave depression in association with a transverse position, more prominent in children than in adults, and occasioning a rather broad contact with the diaphragm. A sort of pseudoaortic configuration results. As compared with a true aortic silhouette, two distinguishing features exist. First, the true aorta configuration in the anterior view reveals an elliptical or parabolic left lower contour in contrast to what has just been described as the *cœur en sabot* type. Second, in the oblique views it shows absence of a right ventricular bulge ventrally and the presence of a left ventricular bulge dorsally. With the *cœur en sabot*, the right ventricle is prominent ventrally while dorsally there is an absence of left ventricular enlargement. The depth of the waist may be spanned by a fainter shadow, as an expression of a hypoplastic pulmonary artery. Because of the torsion of the heart about its horizontal axis, however, a normal sized pulmonary artery trunk is easily concealed; dilatation of the trunk may be indicated by nothing more than a flatly prominent knob, filling the depth of the waist. Half-way between the concave depression and the upper apex, on the left side, a short indentation in the contour is occasionally observed, indicating the interventricular sulcus. The aortic shadow may be prominent, which permits one to state that the aorta is moderately large while the dextroposition itself cannot be diagnosed by means of roentgen rays. The pulsations of the silhouette are very slight. The roentgenological type here described is characteristically observed in the presence of the tetralogy of Fallot.

The other type shows a normal or vertical position of the cardiac shadow. The *cœur en sabot* form is usually preserved. The waist of the left contour has nearly disappeared; in some instances there is a rather large prominence of the conus, easily observed and verified by its ventral position in the right anterior oblique view. The aortic knob itself is well preserved. Pictures as described here are found in cases with closed interventricular septum. This is a relatively rare type.

Finally, it is to be stated that silhouettes may be found without any characteristic features whatever.

The intrapulmonary vessels appear normal or relatively small. This is of especial interest when at the same time the pulmonary conus and the pulmonary artery are considerably enlarged. An unusual vascular distribution

along the medial areas of the lung fields, indicative of the collateral circulation, will perhaps be observed some day on roentgenograms. Stereoscopic technique should be of value.

**Tricuspid Atresia.** Agenesis of the tricuspid orifice is associated with right ventricular hypoplasia, left ventricular hypertrophy and dilatation, and there is generally present a communication between the atria and ventricles. This combination is referred to as Wiedland's disease. The right atrium is dilated. The pulmonary artery may be atretic, in which case the ductus arteriosus remains patent. Cyanosis is present, the E.C.G. invariably shows left axis deviation, and the life expectation is usually but a few months or occasionally a few years. Roentgenologically, enlargement of the heart is noted because left ventricular and right atrial enlargement overcompensate the right ventricular diminution. The waistline of the silhouette is well marked, and the enlargement bears to the left and—when observed in the left anterior oblique view—dorsad-caudad. When the pulmonary artery is narrowed, the vascular pedicle is found to be very small.

**Coarctation of the Aorta.** At a point distal to the origin of the left subclavian artery, very rarely proximal to it, and close to the insertion of the ligamentum arteriosum, there is found an atresia or a stenosis of varying degree. The site of this anomaly varies somewhat. The form varies from a mere diaphragm formation to a small channel or complete interruption a few cm. in length. If the degree of stenosis is sufficiently pronounced, a collateral circulation is established, widened and tortuous intercostal arteries being a part of the picture. This leads to erosions of the caudal edges of the ribs in their dorsal portions, usually quite pronounced in adults although only occasionally demonstrable in childhood. The heart is often found to be normal in size with some left ventricular hypertrophy, or there may be a greater degree of left ventricular dilatation, always present when there is concomitant aortic valvular disease. The size of the aorta varies. The ascending aorta is commonly the seat of dilatation, from a slight degree to formation of an aneurysm. It may be of fairly normal size, or finally a true hypoplasia may be present, especially with concomitant patency of the ductus arteriosus, if the patency is great enough, the right ventricle is enlarged and there is no need for the development of a collateral circulation.

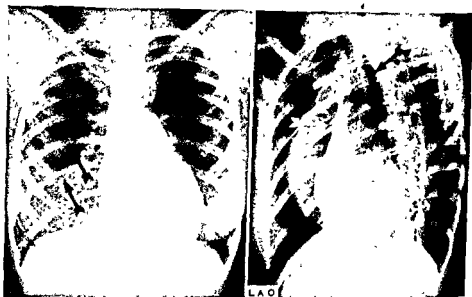


FIG. 310—20 yrs, F. Unable to play in childhood. Marked cyanosis, clubbing, maximum systolic murmur in 2nd and 3rd lt intercostal spaces P<sub>2</sub> easily audible. Pulmonary tuberculosis with cavitation. Rbc 7.5 mil, hb 107%. ECG right axis dev. Ant (and obl) views heart slightly enlarged, nothing else of note. Hilar vessels quite small. Postmortem: infra-valvular pulmonary stenosis, interventricular septal defect, right ventricular hypertrophy. Stenosis of tricuspid orifice (slight). Tuberculosis of lungs. Essentially negative roentgen findings in the presence of a typical congenital cardiovascular malformation.



The clinical diagnosis then will center entirely on the findings of a patent ductus. Distal to the site of the coarctation, the aorta resumes either a normal lumen or is moderately dilated. The region of the stenosis or atresia often shows calcifying atherosclerotic changes.

Coarctation of the aorta must always be thought of whenever high blood pressure is found, especially in young individuals. The subsequent clinical examination reveals that the high pressure is limited to the upper part of the body. Aneurysmal formation in early life should also make one think of



FIGS 311, 312—18 yrs, F Rheumatic process for 1 year Shortness of breath Headache Cardiac enlargement to lt Visible pulsations corresponding to internal mammary arteries and over arterial vessels in back Vessels at rt side of neck pulsate extensively, those at lt side are faintly perceptible BP: rt arm 240/140, lt arm 140/110; femorals not palpable ECG.: nsr, T<sub>1</sub> <sub>2</sub> negative Coarctation of aorta Ant view: Aortic knob visible Enlargement of silhouette to lt Erosions along caudal margins of ribs (bl arrows) Lt. ant. obl. view. aortic arch ends in a knob-like fashion and cannot be traced farther (bl. arrow). Contour of ascending aorta (wh arrow)

coarctation of the aorta as a possibility. Complications caused by high blood pressure and the threat of a rupture of the aorta must be remembered in considering the prognosis.

The erosion of the ribs is the most unequivocal roentgenographic sign, although it is not always present nor is it always very marked. The youngest patient in whom this sign was reported was six years of age. These erosions are multiple, bilateral and limited to the caudal margins of the dorsal portions of the ribs. They are limited to the right hemithorax when the seat of coarctation is proximal to the origin of the left subclavian artery. They are of smooth outline and there is no change in the adjacent bony structure. The dense and tortuous intercostal arteries may be directly observed as serpiginous shadows in the intercostal spaces.

The ascending aorta may appear small, normal in size, dilated, or in the form of an aneurysm extending into the right lung field and ventrad. The dilatation is either actual or dynamic, with the latter finding particularly applying to younger individuals. Forceful pulsations are commonly observed. The aortic knob often marks the site of the constriction and is very frequently, though not always, found to be small or absent. This finding is quite striking in connection with dilatation or aneurysm formation of the ascending aorta and occasions an asymmetrical appearance of the vascular shadow. Its greatest portion is visualized to the right of the midline. In a small minority of cases,



Figs 313, 314—27 yrs, M History of no significance. Complaints of slight headache. Pulsating arteries over scapular and shoulder regions. Apical thrust heaving. B.P. 190/110 in the arms, 130 in the legs. Coarctation of aorta. *Ant view* Prominence of aortic knob. Erosions along caudal margins of ribs (bl arrow). *Lt ant obl view* end of arch of aorta is noted to curve into the very first part of descending thoracic aorta. Here (upper bl arrow) actual narrowing of aorta is noted. (Lower bl arrow) ventral contour of descending aorta. (Wh arrow) left main branch of pulmonary artery.

the aortic knob has a normal appearance or may show an undue prominence. Here it must be assumed either that the site of constriction is more distal than usual, or that an elongation of the aortic arch extends considerably to the left, ventrad to the constriction. The first alternative seems to be the more likely. The barium filled esophagus displays its normal right sided deviation at the level of the aortic arch; the measurement of the aortic arch by this method may reveal small figures. The pulmonic arch often is clearly seen, perhaps because it is not overlapped by the shadow of the descending aorta. The heart is normal in size or is slightly enlarged to the left, with some tendency to aortic configuration, unless this is counteracted by a low position of the diaphragm. Gross enlargement occurs only in the course of associated disease such as aortic regurgitation.

The left anterior oblique view may reveal the second unequivocal diagnostic roengenological sign, namely, a narrowing, or a gap in the aortic arch. This is sometimes impossible to demonstrate because there may be only a diaphragm present which, by projecting into the lumen, causes the constriction while the outer aspect of the vessel is scarcely altered; or the youth of the patient may not permit of adequate visualization of the aortic arch. The direct diagnosis is facilitated by atheroma of the aorta, considerable dilatation of the aorta proximal to the site of the stenosis, and by a marked narrowing of the vessel lumen, particularly if the narrowing is of some length. A flattening or indentation may be observed along the convex contour at the very beginning of the descending aorta. Or again a definite break may be seen in this outline behind which the shadow of the aorta cannot be traced through the shadow of the vertebral column. Or the aortic arch narrows in an almost triangular fashion with its cephalic border clearly pulsating; dorsad to this gap there is neither a definite shadow noted nor are pulsations observed. Where the anterior view discloses a definite prominence of the aortic knob, the left anterior oblique view permits visualization of the aortic arch but the dorsal contour of the proximal portion of the descending aorta swings ventrad and a kink-like narrowing in front of the spine is noted. All these findings are quite impressive in the presence of dilatation of the ascending aorta, for in aortic disease with dilatation, we find either the whole arch sharing in the dilatation or displacement; thus the aortic knob is prominent in the anterior view and the arch reveals a wide swing in the left anterior oblique view, considerably overlapping the shadow of the spine. Contrast visualization has permitted the *direct demonstration of the narrowed aortic segment.*

**Persistent Right-Sided Aortic Arch.** In reptiles the primitive, embryonic, paired aortas persist while in birds the right sided arch alone is maintained. Both of these forms of development may be found in mammals, the latter type much more frequently. When the right or left sided aortic arch persists, the first portion of the ascending aorta remains in normal relationship with the pulmonary artery, but the second portion ascends craniad. It may deviate into the arch just above the angle between the trachea and the right bronchus or it may pursue a longer course craniad so that the arch is not at all in contact with the right bronchus. But under all circumstances the left border of the arch corresponds to the right border of the trachea and esophagus. The right arch has, as a rule, the size of a normal aorta. It may occasionally remain exclusively on the right side, together with a part of the descending thoracic aorta; or it may cross the midline to the left, dorsal to the esophagus, this being more common. At the end of the arch and at the beginning of the descending aorta there is often found a diverticular or ampullar enlargement, retroesophageal in site; this represents the root of the obliterated left dorsal aorta. From this diverticulum are given off the

the anterior aorta and the left subclavian artery. The latter may, however, arise from the arch, at the right side of the trachea, and to the left. Or it may have its origin as the first branch from the arch, or from the left innominate artery, again at the right side of the body, and will cross in front of the trachea to

the left. In addition to the right arch, a left arch may persist, as a rule, it is not a full sized arch but a smaller vascular-fibrous ring, in front of and to the left of the trachea, and is formed by the left subclavian artery, a short occluded vessel and the left descending aortic root. The descending aorta may, in case of tortuosity and elongation, extend considerably into the right chest cavity.

At the level of the right sided retroesophageal aorta and of the previously

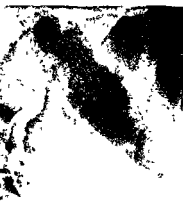


FIGS 315, 316, 317—42 yrs, M Beginning syringomyelia, no complaints as to cardiovascular system. Clinical findings negative, except that basal sounds are louder lt than rt BP 110/75 ECG nsr, lt axis dev, slight degree. *Ant view*: aortic knob noted at rt side, outlined by some lime salt deposits (wh arrow). At this level (short bl arrow) trachea and barium-filled esophagus are noted to deviate to lt, reverse of normal findings. *Rt ant obl view* (A) At level of aortic arch (wh arrow), trachea deviates ventrad. Aortic arch is



RAO

A RAO



B

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mentioned left sided aortic diverticulum, the esophagus and trachea deviate to the left and ventrad, the degree of deviation varies in different cases.

The persistence of the right sided aortic arch occurs as an isolated mal-

formation or, less often, in combination with others. It usually has no clinical significance, although occasionally an esophageal and rarely a tracheal constriction is found. This is produced by an enlargement of the diverticulum, sometimes in connection with atherosclerosis, syphilis or hypertension. It may be caused by the formation of a vascular or fibrovascular ring about the trachea and esophagus, the extreme case being represented by the persistence of both aortic arches; or it may be due to the previously described retroesophageal course of the left subclavian artery, or due to the passage



FIGS. 318, 319—58 yrs, M History: Progressive difficulty in swallowing for past year. Findings: Essentially negative, rate 84/min, B.P. 112/90, E.C.G. Nst, lt axis dev, otherwise normal. Esophagoscopy (Dr C L Jackson) The lumen was somewhat compressed from the rt side at the level of the aortic arch, and here an indurated ulcerated lesion, having the appearance of carcinoma, was encountered.

*Ant. view:* 1, leaf of diaphragm elevated, 2, no definitely clear demarcated aortic knob on lt side, 3, aortic knob on rt side; 4, tortuous descending thoracic aorta (as revealed by rotation), stenosing esophageal lesion (bl arrow). *Rt Ant. Obl. View* 5, aortic arch, 6 trachea, deviated ventrad, 7, esophagus, deviated ventrad, with irregular narrowing below the maximum indentation, 8, dilatation of esophagus, in connection with spastic narrowing at diaphragmatic pinch cock.

*Course.* Death a few months later.

*Comment.* A congenital anomaly, right sided aortic arch, is present. The aorta passes to the right and behind both esophagus and trachea instead of pursuing its normal course in front of these organs. Difficulties in swallowing were not caused by the congenital anomaly in conjunction with the associated deviation of the esophagus, but were due to an intrinsic cause, carcinoma of the esophagus.

of a patent ductus arteriosus to the right. We add that the left carotid artery, and occasionally the left subclavian artery, crosses in front of the trachea. The knowledge of these vascular anomalies is of interest to the clinician, surgeon, esophagoscopist and roentgenologist.

The aorta is also to the right in the presence of a complete transposition of the viscera or in combination with some cases of isolated dextrocardia. But here the embryological development is different. The forward displacement of

the trachea and esophagus is not found and we are dealing with an actual mirror image of the aorta.

The anterior view reveals a well-marked pulsating vascular shadow to the right which extends craniad and slightly outward toward the right sternoclavicular junction. In curving to the left and medially, a more or less pronounced aortic knob is formed whose level is often more craniad than one would expect for a normal left sided aortic knob. Within the right contour another border is often visible, which is parallel with it or at first slightly concave and, farther caudad, slightly convex. This indicates that the descending aorta maintains its course to the right of the spine. The contour below the aortic knob may also reveal a convex bulging into the right lung field. In this case we are dealing with an elongated descending aorta within which the denser ascending aortic shadow may be visualized. Finally, the descending contour may overlap the ascending contour only for a short distance; for instance at the level of the right hilus. If only one right contour exists, it is probable that the descending aorta has crossed to the left. It is not known whether or not the superior vena cava takes part in the formation of the contour. The left contour is either straight, or more often is slightly convex at or just below the level of the right sided aortic knob. In the latter instance, the entire aortic shadow has a club-like appearance. The left sided aortic knob is formed by the diverticulum, i. e., the root of the obliterated left aortic arch, or is due to the persistence of a left sided vascular ring. Both the trachea and the esophagus, the latter revealed by barium paste, deviate to the left at the level of the right sided aortic knob, sometimes only slightly, although more often in a quite pronounced manner. A second deviation of the esophagus, to the right and more caudad, may be observed in case of tortuosity of the descending aorta.

The right anterior oblique view shows the visible portion of the aortic shadow in front of the trachea to be quite small; when well marked, a diagnosis of a pretracheal vascular ring should be made. The trachea and esophagus, at this level, deviate ventrad, sometimes in the form of a rather wide arch, sometimes more flatly and only for a short distance. Rarely there may be an additional small indentation along the dorsal contour of the esophagus which is probably caused by the left subclavian artery. Dorsad to the trachea and esophagus there is noticed a round shadow mass which represents the aortic arch. Within it there may be a denser and smaller round area; this corresponds to the diverticulum. The displacement of the trachea and esophagus is apparently due, primarily, to this diverticulum and therefore varies with its size; as a matter of fact, the displacement, referring to the

along the dorsal contour of the barium-filled esophagus. Another ventral displacement of the esophagus is occasionally noted near the diaphragm if it is crossed by an elongated descending aorta.

In the left anterior oblique view, one notes a rather large aortic shadow in front of the trachea. Both trachea and esophagus either deviate ventrad at this level, or, less often, hold an almost straight course. The swing of the



Figs. 320, 321—6 wks., F. *History:* A moderate degree of cyanosis was present since birth, and became exaggerated at feedings and while crying. *Findings:* Cyanosis was present, the heart was enlarged, there were no murmurs, and the rate was 156/min. The hb. was 18 gms and the rbc 5 mill. ECG: Sr, P<sub>1</sub>, slightly prominent, rt axis dev.

*Post view* (age 17 days) The cardiac silhouette is moderately enlarged and somewhat globular in shape. The vascular pedicle is short and quite small. *Lat view* (illustration omitted) The mass of the heart lies ventrad.

*Course:* Cyanosis persisted, respiration was rapid. No heart murmur heard at any time. Death.

*Postmortem* (age 39 days) The heart was enlarged. The base-apex measurement was 6 cm., and the diameter at the base was 4.2 cm. The ascending aorta was noted in front with pulmonary artery situated farther dorsad and but slightly to the lt. The weight of the heart was 40 gms. The rt atrium was considerably dilated and showed some hypertrophy of its wall, the lt atrium showed questionable dilatation and normal wall thickness. The foramen ovale was patent, the opening measuring 0.6 cm in diameter. The ductus arteriosus showed evidence of recent closure. The rt sided a-v orifice showed two leaflets, but there were three leaflets at the lt side. Both the rt and lt ventricular walls measured 0.9 cm in thickness. The rt ventricular cavity was slightly dilated and opened directly into the aorta. The lt ventricular cavity was slightly dilated and opened into the pulmonary artery. The aorta was slightly dilated and opened into the pulmonary artery. There was atelectasis, congestion, and patchy hemorrhages.

*Comment:* Aorta and pulmonary artery were completely transposed. There was also

patent, but the direction of flow is conjectural. The narrowness of the vascular shadow in the roentgenogram is due to the position of the pulmonary artery which was chiefly dorsad to the ascending aorta. For the same reason we do not note the presence of a pulmonary arch proper.

The aortic arch behind the trachea, as observed in normal cases, is absent here; however, a considerable shadow mass may be observed in the presence of an unusually large diverticulum or of a second, left sided aortic arch.

A constriction of the esophagus from behind, without deviation, was ob-

served in a child; the postmortem study revealed the presence of a double aortic arch with ring formation.

The absence of a left-sided aortic knob is also observed in the following instances: coarctation of the aorta, hypoplasia of the aorta as in combination with an interatrial septal defect, certain types of transposition of the large vessels, persistence of a left-sided superior vena cava, and a mirror type right-sided aortic arch (see above). The deviation of the trachea and esophagus to the left and ventrad has often been incorrectly interpreted, an aberrant thyroid, an aneurysm, or a mediastinal glandular mass were considered to be present, and in at least one case a mediastinotomy was done.

If one suspects the presence of a right sided aortic arch, one should proceed to take roentgenograms in the three standard positions, first without and then with barium filling the esophagus.

An unusual prominence of the aortic shadow to the right is observed with two different associated conditions: 1) syphilis, which affects the ascending aorta; 2) excessive elongation and tortuosity of the descending aorta. Concerning the second condition, it will be recalled that the descending limb of the normal aorta lies in the left vertebral gutter, and when it elongates it will deviate in the form of an arc toward the left lung. In the presence of the anomaly under discussion when the descending limb is chiefly situated to the right of the spine, and when it undergoes elongation and tortuosity, it will develop prominently into the right lung field, tending to pull the esophagus with it. This is best observed by rotating the patient into the right anterior oblique view.

**Transposition of the Great Arterial Trunks.** In this condition there is an alteration in the position of the two great vessels relative to the ventricles of the heart or to each other at their origins. Many forms in combination with other anomalies may exist. Only two types will be mentioned: one in which both vessels arise from the right ventricle; the other in which they arise from the reversed ventricles or from one common



FIG. 322—3 mos. M. Paroxysmal attacks dyspnea, pallor, sweating. Cyanosis on crying vigorously. Cardiac enlargement, normal heart sounds, ECG n.s.r., low voltage QRS, T<sub>1</sub>, <sub>2</sub>, <sub>3</sub> negative. Ant. view enlargement of silhouette, especially to lt. Postmortem heart weight 91 gm (no av 25 gr). Hypertrophy and dilatation of lt. ventricle, valves normal. Lt. coronary artery originates from pulmonary artery. Fibrosis between muscle fibers of lt. ventricle, slight Cardiac enlargement due to inadequate nourishment of ventricular walls: abnormal origin of left coronary artery. Congenital malformation. From Bland, E. F., White, P. D., Garland, J., *Am Heart Journ*, 1933, 8, 787. Courtesy Dr. G. W. Holmes, Mass. Gen'l Hospital, Boston, and C. V. Mosby Co., St. Louis.



ventricle Both aorta and pulmonary artery are either normal in size or enlarged. They are situated either side by side or the aorta is ventrad to the pulmonary artery. In all events the aorta lies further to the right, and the pulmonary artery further to the left than in the normal heart. The heart is always enlarged. A persistent and sometimes extreme cyanosis is present and,



FIG 323—2 mos, F Failure to gain weight Slight cyanosis Cardiac enlargement Systolic rumble over heart Normal Rbc count. *Ant view.* large spherical silhouette Vascular shadow very short Fluoroscopy: pumping pulsations at either cardiac border *Rt lat view* trachea displaced (bl arrow) Both roentgenograms taken at inspiration. Postmortem: almost the entire anterior surface is formed by the ventricular mass, the greatest portion by the lt. ventricle, the small rt ventricle forming the rt border One atrium posteriorly, partly subdivided Pulmonary artery twice as large as the aorta, these vessels in normal relation to

no bulge in the region of the inf v cava (inferior posterior recess of pericardial cavity) An example of tracheal displacement in the presence of a large heart with congenital malformations

although in a few instances adult life is reached, death usually occurs in infancy.

The roentgenologic appearance varies. One notes a large heart shadow, often globular in shape and quite similar to that in the presence of a pericardial effusion. The enlargement of the right ventricle—since this cavity pumps blood through the systemic circulation—is noted in the left anterior oblique and the lateral views. The vascular shadow may be extremely small, especially in infants; in such cases, the pulmonary artery is found dorsad to

the aorta. The considerable cephalic enlargement of the heart accounts in part for the inconspicuous size of the vascular shadow. In other cases, however, the vascular shadow is wide, due to a superimposed thymic shadow, a much enlarged right auricular appendage or actually large vessels. As far as deductions can be drawn from the few available observations, one may state that a prominence of the pulmonic arch and of the conus area of the right ventricle are absent. This is especially striking when there is a considerable associated enlargement of the intrapulmonary branches.

**Left-Sided Origin of the Right Subclavian Artery.** The right subclavian artery may arise as the last vessel from the aortic arch and, in order to reach its extremity, crosses the midline. In four-fifths of the cases it is situated between the esophagus and the spine and, because of its diverticular-shaped origin, swallowing difficulties may arise from pressure against the esophagus (dysphagia lusoria). On filling the esophagus with barium paste a semicircular indentation is noted along its left and dorsal circumference, the level of which exactly corresponds to the top of the aortic arch. Rhythmic systolic movements may be noted along this bend, and this finding serves to exclude a lymph- or thyroid-node as cause for this indentation. Caudad to this bend the aortic arch deviation of the esophagus proper is poorly marked.

**Anomalies of the Large Veins.** They are easily compatible with life. In rare instances the right superior vena cava is absent while the left persists. Here the roentgenogram reveals, even in childhood, an unusual visibility of the ascending aorta. More common is the persistence of both superior venae cavae. Here the vascular shadow is widened and no details are visible in the region of the aortic knob and pulmonic arch. The pulmonic veins rarely empty into such a persistent left superior vena cava. When they do so, the result is much enlargement of the right heart while the left heart and the aorta remain small. The duration of life is very short.

**Other Rare Conditions.** General cardiac enlargement is found in the presence of a persistent truncus communis or solitarius and with the anomaly known as bilocular or trilocular heart. An anomalous origin of the coronary arteries has been observed together with cardiac enlargement. An aortic configuration is associated with subaortic stenosis. Roentgenologic evidence of aortic regurgitation may indicate that there has been endocarditic damage to the bicuspid aortic cusps or that a congenital aneurysm of the wall of the right sinus of Valsalva has ruptured into the right ventricle. Pulmonary regurgitation may be isolated, *f.i.*, in association with bicuspid pulmonic valves, or combined with stenosis of the orifice or other anomalies, here marked expansile and collapsing pulsations are observed along the widened trunk and branches. (This is dealt with in detail in chapter X).

**TRACHEAL DISPLACEMENT** This has previously been referred to in this chapter. Sometimes the cause is not clearly established. In order to evaluate such a displacement properly, roentgenograms should be made in the anterior and lateral views in both the inspiratory and expiratory phases. In the anterior view there is a marked buckling and deviation to the right at the height of the expiratory phase, and in the lateral view there is some buckling associated with moderate diminution in the caliber of its lumen. Tracheal displacement

has therefore pathognomonic significance only when present in the inspiratory phase. A markedly enlarged thymus gland also causes such a displacement.

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## CHAPTER XIII

### DISORDERS OF CARDIAC RHYTHM, RATE, CONTRACTION

The roentgenologic method plays a comparatively insignificant rôle here

**PAROXYSMAL AURICULAR TACHYCARDIA.** *The reaction of the heart size to such attacks depends upon the condition of the heart muscle and possibly also upon the duration of the attack. Short attacks will produce in a grossly healthy heart either no changes or a slight tendency to a decrease in its silhouette. A prolonged attack may cause congestive failure in the greater circulation, and the shape and size of the silhouette will undergo changes due to the engorgement of the liver. Right sided dilatation also may take place. Dyspnea, as a rule, is absent and one notes, roentgenologically as well as clinically, absence of pulmonary congestion. In patients in whom dyspnea and even pulmonary edema occurs, one does find, however, the roentgenologic equivalents for failure in the lesser circulation, but here an associated condition, for instance hypertensive cardiovascular disease, is present. Diseased hearts seem to undergo more rapid enlargement but serial studies do not exist in which exact criteria for comparison are available. The amplitude of the pulsations diminishes. In a case with auricular flutter, it was observed, kymographically, to decrease not more than 12% as a maximum. Paroxysmal rapid heart action in young infants may be overlooked. When it lasts for several days it may cause pulmonary congestion. The resulting presence of dyspnea, cyanosis, fever, leucocytosis and diffuse fine mottling in the lung fields—most marked medially—may readily suggest the erroneous diagnosis of pneumonia. Nodal tachycardia has been differentiated from sinus and auricular tachycardia by observing the characteristic simultaneous movement of each border of the silhouette.*

By sewing wire electrodes to the right atrium in dogs and by applying a certain type of electric current it is possible to produce regular tachycardias

the size of the cardiac silhouette in the heart rate was likewise obtained by giving atropin to morphinized dogs or by applying electrical stimuli to one of the vagi following double vagotomy. The size of the silhouette reached a minimum at the rate of 200 per minute, it was thought that the fall in venous pressure was responsible because, when it was kept constant by infusion, no changes in the silhouette took place for rates between 60-210.

**AURICULAR FIBRILLATION AND FLUTTER.** Fluoroscopy cannot detect fibrillation; it reveals only the response of the ventricles by complete irregularity in the time of their movements and by continuous changes in the size of

the cardiac silhouette. The absence of auricular waves, but careful observing. It is not known during such attacks.

While the size of the left atrium is not directly related to the presence of

auricular fibrillation, yet in the presence of great enlargement of the left atrium auricular fibrillation is invariably present. When the onset of auricular fibrillation coincides with the development of cardiac enlargement, evidence of coincidental cardiac failure will be observed. And in the non-rheumatic group when the left atrium is definitely enlarged, auricular fibrillation is uniformly accompanied by failure. In attempting to restore sinus rhythm by the use of quinidine sulphate, it is well to remember that such an attempt is likely to fail in the presence of even a moderately enlarged heart.

There are patients who have auricular fibrillation, transient or permanent, without evidence of thyrotoxicosis or organic heart disease. Here a normal heart size is present and remains unchanged over long periods of time.

Some auscultatory findings of mitral stenosis are less characteristic when auricular fibrillation supervenes. Thus the roentgenological data are welcome to the clinician.

With the same method as that mentioned above, auricular fibrillation was produced in dogs, the silhouette areas revealed either no changes or they were found to be increased.

Auricular tachycardia due to auricular flutter may occasionally be observed by placing the patient in the right anterior oblique view and visualizing the rapid atrial action as transmitted to the barium-filled esophagus. Kymographic tracings show the flutter waves superimposed upon the tracings obtained from the right cardiac and superior vena cava silhouette borders.

**BRADYCARDIA, NODAL RHYTHM AND COMPLETE ATRIO-VENTRICULAR DISSOCIATION.** One notices large pulsatory amplitudes in all instances of *bradycardia*. This is an expression of the increased stroke volume (plus 40-50% in cases of complete heart block). With very slow ventricular rates this amplitude may increase up to

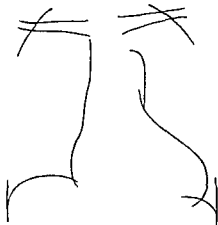


FIG 324—40 yrs, M Diphtheria in childhood. Known to have slow pulse for 13 years. Attacks of dizziness, dyspnea on effort. Heart moderately enlarged, systolic murmur everywhere. Rate 40 B P 110/60. ECG complete atrio-ventricular dissociation. Complete block of long duration, etiology unknown. *Ant view* (diastolic silhouette): moderate enlargement, L 15.4, B 9.6, T 16.0, Th 23.2. Lung fields clear. Fluoroscopic amplitude of pulsations: lt lower contour 0.9 cm, rt lower contour 0.7 cm, aortic knob 0.5 cm. The more rapid auricular action can be differentiated from the slower ventricular action. Rt ant obl view (illustration omitted). It atrium not enlarged. An example of cardiac enlargement in the presence of a heartblock of long duration.

rhythm even after cases with essential hypertension, congenital malformation, valvular lesions and coronary artery disease are excluded as far as possible. The cardiac area is noted to increase 5-20% when bradycardia is induced by means of neosynephrin hydrochloride. The slow ventricular rate is accompanied by an increased excursion of the amplitude of the aorta and pulmonary artery arch and increased pulsations of the hilar branches of the pulmonary artery. The more marked the bradycardia the more marked is the relative and absolute change in the size of the cardiovascular silhouette; the heart shadow itself appears as a pumping sphere.

This movement to and from an imaginary center is most uniform in the presence of the *nodal rhythm*; here the atria and ventricles pulsate simultaneously or almost so. The extent of excursion is equal at both sides or greater at the right. With each cardiac contraction there is noted a sudden lateral movement of and increase in that shadow area which corresponds to the superior vena cava. On account of the simultaneous contraction of the right atrium and ventricle, blood is actually thrown back into the superior vena cava. The excursion at the right border of the vascular shadow is greater than at the left side (aorta). Marked systolic pulsations of the superior vena cava are also observed in the presence of tricuspid and aortic valvular regurgitation; as a rule, however, bradycardia is not found with these conditions. With aortic regurgitation, the aortic knob shows a greater extent of excursion than does the cranial right vascular border.

In *complete atrio-ventricular dissociation* the excursions of the atria can be seen to take place at a higher speed than the ventricular mass and independent of it. Provided there is no auricular fibrillation present, a fluttering, superimposed movement is noted, quite easily visible at the right lower silhouette border and often, though not always, in the region of the left auricular appendage. The movement of the right border changes continuously in type, because ventricular and atrial action constantly interfere. The lateral movement of the left lower contour is occasionally seen to be superimposed upon by another, *extra movement*. This can be demonstrated by kymography also; it apparently corresponds in time to an atrial systole which causes an impact of blood against the relaxed ventricular wall. Following exercise, the rate of the atria is seen to increase considerably while the rate of the ventricles, as a rule, is but little changed. If there is a constant ratio between auricular and ventricular rate, for instance with the *2:1 block*, a simpler type of pulsation is observed. In the left auricular appendage region there is one additional movement noted during the ventricular diastolic phase. The right border shows small excursions which are superimposed in regular sequence over the slow and larger ones.

**PREMATURE BEATS.** They are characterized by a small excursion which is followed by a slow and wide lateral movement and a correspondingly large medial movement. The aortic shadow shows either a small excursion or none at all (*frustrated ventricular contraction*), while the beat following the pause causes a large excursion. Kymographic studies in premature ventricular beats have shown that contractions which are recognizable only at the apical area are always frustrated, that is, they are not shown in the kymogram of the aorta. If the movement is present over the cranial portions, the result of the

contraction, though it may be small, is always recognizable in the kymogram of the aorta.

**ALTERNATING PULSE.** Fluoroscopy and kymographic tracing reveal alternating excursions along the cranial portion of the left lower contour, every other systolic movement being diminished. These studies have not helped to decide whether the mechanism of alternation is partial or total alternating hyposystole or primacy of certain hemodynamic conditions.

**BUNDLE-BRANCH BLOCK.** The movements of the aortic knob and of the pulmonic arch have been registered kymographically in instances of the so-called common type of bundle-branch block, which is characterized in the ECG by a widening of the initial deflexion to 0.14 sec or more, its upward direction in lead I and downward direction in lead III, with the respective T waves in reverse direction. Measurements of the interval between the beginning of the initial deflexion and the beginning of the pulse wave in the aorta and pulmonary artery revealed that the aortic pulse fell much later than the pulmonic, i.e., a delay in the ejection from the left ventricle was to be postulated. An asynchronism of the same order of magnitude was also noted in patients with split first sound but without intraventricular conduction defect. A bundle-block lesion is commonly, though not always, associated with cardiac enlargement, and this holds true also for cases where there is no hypertension.

Intrathoracic, especially mediastino-pericardial pathology, as revealed by the roentgen ray study, may account for the clinical finding of a *paradoxical pulse*.

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## CHAPTER XIV

### PERIPHERAL VASCULAR DISEASE

(The reader is referred to remarks in chapters I, III, V.) The clinical diagnosis of peripheral vascular disease depends largely on indirect methods such as palpation, study of the response to postural and temperature changes and oscillometric determination. The last named method will determine the upper level of an arterial obliteration but renders no information as to the mode of circulation below the obliteration. The clinical course, for instance, is entirely different should there be a segmental obliteration in the superficial femoral artery or should the process extend into the posterior tibial; rapid gangrene will follow in the latter instance.

**INDICATIONS FOR VASOGRAPHY.** The functional condition of a vessel and of the tissues supplied by it depends mainly upon the size of the vessel's lumen. Vasography permits the study of this lumen. The roentgenogram may demonstrate calcification in vessel walls; but there is no close relationship between this finding and the actual size of the lumen. Calcification may also indicate a straight course of the vessel while the injection reveals a thread-like tortuosity of the lumen. Vasography may demonstrate: 1) congenital variations; 2) alterations of the caliber and of the character of the wall changes, including size of the involved area. For practical purposes there are to be considered: 1) determination of location of emboli and thrombi; 2) the study of the site of vascular occlusion and of collateral circulation to determine whether conservative operations like sympathectomy or segmental arteriectomy are worthwhile or whether an amputation is necessary. In the latter instance information is obtained as to which of the collateral vessels might be saved at the operation, 3) the demonstration of vascular aneurysm of traumatic, arteriosclerotic or congenital etiology; 4) a differentiation between spastic and organic vascular disease; 5) study of the extent of varicosities, 6) study of the vascular supply in tumors and inflammatory disease.

One important difficulty in vasography must not be lost sight of, that it is impossible to demonstrate in a roentgenogram, or even in a series of them, the complete vascular tree on either the arterial or the venous side.

**EVALUATION OF CONTRAST MEDIA.** Previously, in chapter I, the different contrast media were mentioned and it was stated that none of them is entirely considered congenitally; it may have temporary ed in the reticulo-damage, although 1/3 of that used

for hepato- and lienography (75 cm<sup>4</sup>). Its chemical effect on the vessel wall is nil. The results of animal experimentations are not uniform. The liver of rabbits develops changes varying from simple cloudy swelling to profound necrosis, depending upon the dose. These changes are followed by fibrous tissue proliferation which gives a picture similar to mild nodular hyperplastic cirrhosis. The bone marrow shows hematopoietic depression. In contrast

to this, the organs of dogs reveal very little alterations. In another series of experiments the superior vena cava and aorta of rabbits were examined 1-160 days following injection into the ear vein. Deposits of thorotrast were noted in the intima undergoing epithelialization, thus causing degeneration of the elastic tissues of the vessels' walls. Arteriography of the brain with thorotrast in man was shown to be followed by the formation of small encephalomalacias with ring-like hemorrhages. Uroselectan and abrodil were shown not to produce changes in the intima when injected into the normal vessels of dogs. Since both substances cause definite vascular pain when injected, one might rightly think that their injection is contraindicated in the presence of vascular disease which is of sufficient severity to produce gangrene, and untoward results have actually been observed, though only in a few cases. Pain, ecchymoses, pallor, cyanosis and coldness, disturbance in motor and sensory functions and even subsequent gangrene have been reported. It is perhaps wiser not to inject these substances whenever tendency to vasomotor spasm is present. Per-Abrodil (diodrast) is said not to cause pain. One should be guarded in accepting statements that the intra-arterial injection of iodine compounds have led to persistent improvement in certain cases of arteritis. The vacillating course of arterial disease and its tendency to spontaneous remissions should be remembered. Sodium iodide is definitely contraindicated on account of the severe reactions which have commonly been observed. The same applies to lipiodol which acts as a foreign body. Biopsies have shown that it embolizes into the capillaries. Injected in amounts of over 5-6 cm<sup>3</sup>. in the presence of vascular disease, it produces circulatory occlusion, aggravation of ischemia with disappearance of oscillometric pulsations, and swelling and a fine purpura may result. In addition another disadvantage has been observed. The oil does not enter all branches, but shows a preference for certain pathways; for instance it favors the superficial femoral artery to the profunda, and the posterior tibial to the anterior.

When contrast media are used, a delayed exposure may be responsible for erroneous film interpretation. With the use of the higher viscous thorotrast especially, the smaller arterial vessels empty more slowly than the larger ones and therefore may be visualized proximal to the column of the contrast medium in the larger vessels. If enough time elapses, the venous channels may also or even exclusively be visualized.

**EMBOLIC OCCLUSION.** The contrast medium ends suddenly in a crescent-like column. The site of an embolus or of a thrombus may be along the wall. In that case the lumen of the vessel appears suddenly narrowed in an eccentric fashion. If an embolus is not found by vasography, it is well to remember that its site may be proximal to that which is suspected. The artery should be clamped and the contrast medium injected in a retrograde direction, proximal to the site of clamping. Preoperative exact location of an embolus permits its removal under local anesthesia while the search for it with extensive dissection of the vessel requires general anesthesia. In this connection one should remember that many patients with embolic occlusion have primary heart disease in which general anesthesia may be contraindicated. It should be realized that an embolus in an otherwise healthy artery is an indication for



FIG. 325—Arteriography. Clinical diagnosis: embolic occlusion of femoral artery. *Post. view*: sudden stop of contrast medium is visualized (wh. arrows). Course: embolus, followed by amputation of leg. Courtesy Prof. Dr. M. Sgalitzer, I. Chir. Univ. Klin. (Prof. Dr. E. Ranzi), Vienna. From Roesler, H., in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System; ed. by Ross Golden, 1936, p. 259. Courtesy Th. Nelson & Sons, New York.



FIG. 326—60 yrs., M. Diabetes diagnosed at age 41. Pain in foot for past 8 months. Slight improvement following periarterial sympathectomy. Gangrene of toes for 3 months, improved by diet and insulin. Pulsations in femoral artery just perceptible, absent in popliteal, posterior tibial and dorsalis pedis arteries. Wassermann negative. Arteriography (Uroselectan). It femur region. *Post. view*: marked irregularity of filling with complete defect in lower third (between bl. arrows), with bridging over by collaterals. Course: amputation in middle third of femur. Microscopic findings: extreme degree calcification in media, thickening of intima, old thrombi.

surgical removal, but the same procedure attempted on a thrombus in a diseased vessel usually ends in failure. Hence a preoperative study of the appearance of the arterial wall in the roentgenogram will be of value.



FIG 327—49 yrs M. Several amputations of both legs because of gangrene, at age 35 and 40, respectively. Ulcers developed on second, third and fourth fingers of rt hand at age 40. Persistent aching in rt hand and forearm since that time and in lt hand and forearm since age 43. When rt hand was opened following tight clenching and simultaneous occlusion of ulnar or radial arteries, color returned in abnormally slow manner. Wassermann negative.

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partly recanalized. From Demel, R., Sgalitzer, M., Kollert, V., *Mitt. Grenzgeb. Med. Chir.*, 1931, 42, 357. Courtesy Prof. Dr. M. Sgalitzer, I. Chir. Univ. Klin. (Prof. Dr. E. Ranzi), Vienna and G. Fischer, publisher, Jena.

Sudden massive *venous thrombosis* is followed by marked spastic arterial constriction and the resulting clinical picture is similar to or almost identical with embolic arterial occlusion. Arteriography is of diagnostic value in such instances.

**ATHEROSCLEROSIS AND THROMBO-ANGIITIS OBLITERANS** A typical vasographic picture of the *atherosclerotic vessel* with marked intimal changes as observed in the old and in the diabetic is characterized by filling defects and plate-like prominences. The vascular outline is irregular, tortuous, sometimes threadlike, and the axis of the vessel seems to have undergone an actual torsion and even angulation. Where a sudden narrowing or constriction is observed in the course of the main vessel, the proximal lateral branches are usually well seen. The number of smaller vessels is often diminished and they may follow a spiral course and occasionally show miliary aneurysms. The fine branches which arise directly from the main trunks are not visualized. The process often reveals a segmental character. The following arteries are sites of predilection: superficial femoral, popliteal and posterior tibial. As compared with senile atherosclerosis of the same occlusive type, the presence of diabetes favors much more marked trophic disturbances.

The process of *thrombo-angiitis obliterans* goes through several phases. First one notices a concentric diminution of the lumen, often segmental in type. Later on a shaggy and moth-eaten appearance is noted and the lumen is greatly reduced in size and pursues an irregular course. The presence of associated atherosclerotic intimal changes cannot be excluded. In the third stage, complete occlusion of the artery has occurred. Collateral arteries, which are not usually seen in the normal arteriogram, become abundant and of considerable size. Such collateral arteries have an irregular, turning and winding course, vary markedly in size in any selected area, cross and recross and often reveal a lateral course. *Thrombo-angiitis obliterans* frequently involves all four extremities. Various stages of the disease may be present in various arterial branches in the same limb. Within one artery the distribution of the lesion is patchy in character; never is the whole artery uniformly altered.

**RAYNAUD'S DISEASE** The digital arteries usually are not filled normally in arteriograms; the distal part shows absence of filling, and the caliber is diminished. One never observes evidence of occlusive arterial disease of the type seen in thrombo-angiitis obliterans, and this aids in differentiating the Raynaud syndrome from cases with vasomotor crises initiating an early arteritis. Since the injection of the opaque material is made into the brachial artery, the question may come up whether an incomplete filling of the distal arteries is actual or attributable to poor technique. This can be decided by determining the proximal point of radiopacity of the artery. If it is noted to be within 15 cm. of the wrist, the digital arteries had ample opportunity to fill completely. In order for filling defects to be significant of disease, they must be constant on all roentgenograms.

**ANEURYSM.** Most frequently the popliteal artery is the site of aneurysm formation. Trauma being an important etiologic factor, location may of course be anywhere. The method of choice for demonstration is the injection of contrast medium directly into the sac, but failure may result when it is

rapidly carried away by a communicating vein or when the injection is attempted into thrombotic material. It is always indicated to attempt a preoperative demonstration of the collateral circulation in the vicinity of the sac. When the circulation is found to be poor, operation should be delayed until measures have been taken to improve it.

**ARTERIO-VENOUS ANASTOMOSIS.** The clinical diagnosis of the acquired, traumatic variety is not difficult. Abnormal congenital communications should be suspected in the presence of varicosities which appear early in life, particularly when of the unilateral type and of unusual locations, in chronic ulcers of the leg in young subjects without obvious cause, and in instances of hemihypertrophy. Arteriography may determine the site and size of fistula(s) but if the anastomosis is large, the arteries cannot be demonstrated. Following resection and suture, the condition of the vessels may be studied roentgenographically.

**BLOOD SUPPLY.** It is well to keep in mind that arteriography gives no information as to the condition of the arterioles, nor does it yield any information as to the state of activity of the vascular process. In other words, no conclusions can be drawn from it regarding progression of the disease. The prime importance of the clinical examination is evident in order to determine the proper level for an amputation, and especially important is the history concerning presence or absence of pain. However, the level of ischemia in a sudden occlusion is quite variable, ranging from 10 to 25 cm. below the site of occlusion. In a slowly progressing occlusion the blood supply to the tissues may be sufficiently provided by the collateral circulation, but the oscillometric reading may fall to zero because the stenosis interferes with the progression of the pulse wave. Arterial disease of the extremities often reveals multiple areas of occlusion, and they are frequently situated farther proximal than one might anticipate. A partial arteriography is therefore an incomplete study and may lead to oversight of a significant localization of the disease process. Gangrene may start in a toe and yet the site of occlusion be far proximal.

To cite examples, following trauma, a foot gradually becomes cold, cyanotic and no oscillometric readings are registered. Arteriography discloses an occlusion of the posterior tibial artery with a fair supply to the heel, and at operation the region of the calcaneus can be saved. A red, non-pulsating, fluctuant swelling is observed in the popliteal region and pulsations in the leg are absent. A cold abscess, tumor or aneurysm is suspected. Arteriography reveals a sacculated aneurysm, and it also indicates the collaterals which must be saved at operation. A case of a diabetic has been reported in whom a skin lesion was thought to be of vascular origin. Arteriography showed a good blood supply and subsequent study proved it to be a traumatic lesion as a result of loss of pain sensation. Another patient developed intensive pain, swelling and cyanosis in one leg. Arteriography from the femoral triangle revealed normal conditions. The diagnosis of thrombosis of the iliac vein was made and verified by laparotomy. An arteriogram may reveal objective evidence for pain and circulatory disturbances that follow trauma to an extremity. Thus a patient, who may have been suspected of malingering, is freed from such suspicion.



Arteriotomy is occasionally used as a therapeutic measure in obliterative vascular disease. Provided that arteriography shows the process to be limited to the femoral artery, there is no contraindication for this operation, but the presence of the process in the popliteal or tibio-peroneal trunk represents a contraindication since important muscular arterial branches would have to be sacrificed.

Caution, of course, is needed in the interpretation of the findings. The visible collateral blood supply may seem to justify a lower amputation, but though it is just sufficient for nutrition, it may be inadequate for the process of sound healing. A case has been reported where insufficient or no filling was visualized at the time of a first injection; later, however, a normal filling was found. Thus, spastic influences must be considered. The arteriogram of a patient with plexus paresis of 24 yrs duration showed a stop in the interosseal artery. The arm had to be amputated. Another contrast medium injection into the brachial artery of the amputated extremity again revealed a stop, but one that was further distad. On dissection the lumen of the artery was found to be free. It had been a functional stop due to loss of muscle movements and to a diminished need for blood supply. Further examples

(Uroselectan), rt lower extremity. Lat view: filling of aneurysmal sac is concave indentation at distal aspect probably caused by thrombus. Size of aneurysm outlined by collateral circulation around it. Course gangrene of foot. Postmortem syphilitic aortitis, atherosclerosis of coronary arteries with old myomalacia. Aneurysms of both popliteal arteries. From Demel, R., Sgaltzer, M., Kollert, V., *Mitt. Grenzgeb. Med. Chir.*, 1931, 42, 357. Courtesy Prof. Dr. M. Sgaltzer, I. Chir. Univ. Klin. (Prof. Dr. E. Ranzi), Vienna and G. Fischer, publisher, Jena.

may serve to show the manifold variations of anatomical processes which complicate interpretation. Passage of the contrast medium does not exclude the presence of a thrombus which has been canalized. In a case of beginning gangrene of the foot, aortography showed a high degree of stenosis of the iliac artery while distal to it no further vascular disease was seen. The usual injection into the femoral artery perhaps would have led one to exclude vascular pathology.

Arterial injection with certain Iodine compounds necessitates anesthesia, general or spinal. When spinal anesthesia is used, slowing of the circulation and vascular dilatation is observed.

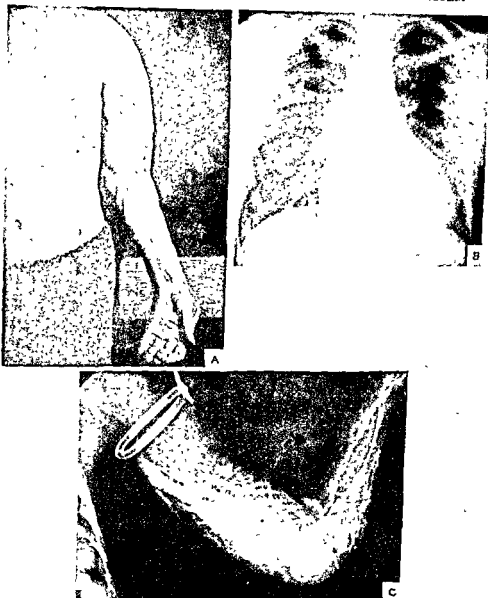
Visualization of the vascular system, as carried out in animal experiments, has increased our knowledge of certain neurogenic, pharmacologic and humoral influences on the blood vessels.

The neurogenic influence on the vascular tonus is well demonstrated in the following experiments on dogs: 1) lumbar sympathetic ganglionectomy and ramisectomy were performed. Weeks or months afterwards the animals were sacrificed. The arterial tree of the hind legs was injected with a contrast medium and vasodilatation was shown to exist. 2) the terminal aorta with all main trunks was resected, along with the lumbar and sympathetic chains. Roentgen examination one month later demonstrated a good vascular supply to the hind legs. If the nerve supply had not been cut in this operation, the dogs would have died with cyanotic and edematous hind legs, and subsequent roentgen examination would have shown scarcely any filling of the arteries. 3) section of the vasodepressing carotid sinus nerve was performed. That this is followed by arteriolar vasoconstriction was demonstrated by means of thorotrast injections. the larger arterial branches were but little



FIG 329—Venography Varicosities Leg Lat view, characteristic longitudinal and transverse distension of diseased veins. Courtesy Prof. Dr. M. Sgaltzer, I. Chir. Univ. Klin. (Prof. Dr. E. Ranzi), Vienna. From Roesler, H., in *Diagnostic Roentgenology*, Nelson's Loose-Leaf System; ed. by Ross Golden, 1936, p. 259. Courtesy The Nelson & Sons, New York.





FIGS 330, 331, 332—19 yrs. M History Hospitalized in connection with bone disease

cortex and irregular trabeculations. A cutaneous pigmentation was noted on the back. The superficial veins of the lt arm were tremendously dilated and tortuous (photograph, A), the larger ones showing systolic pulsations, and a thrill was palpable throughout. Increased pulsations with bruit and thrill were noted over the larger arteries above the lt clavicle, along the lt arm and below the lt inguinal ligament. The heart was definitely enlarged with the apical thrust very forceful. A harsh systolic murmur was heard over the aortic area. On compression of the lt. axillary or femoral arteries, the heart rate slowed from 80 to 60 per min and the cardiac murmur diminished perceptibly. This

modified while there was an almost complete disappearance of the small arterial ramifications.

When thorotrast is injected into an extremity of an anesthetized dog, the size of the arteries can be accurately determined. When, at another time, the arteriogram is done after the main artery has been compressed for a few minutes and then released, the whole arterial tree shows an intense vasodilatation that continues from a few minutes to half an hour. The degree of this temporary vasodilatation equals the one noted as a permanent feature in animals in which the lumbar sympathetic chain and ganglia had been removed. Venous compression will reveal constriction of the large arterial vessels while all the small branches appear much dilated. These findings are of interest in view of the alternating arterial or venous compression that is used in the treatment of peripheral arterial disease.

When one-quarter of a rabbit's blood is removed by cardiac puncture and an equal amount of thorotrast is injected into one of the ear veins, all vessels with a diameter greater than 0.02 cm. are visualized. The rabbits behave normally during the first hour but die a few hours later. Experiments that are carried out within a few minutes following contrast visualization are therefore valid. The intravenous injection of 2 cm<sup>3</sup> of histamine is lethal for the rabbit within 4 min., causes a decrease in volume of the systemic and pulmonic arteries, dilatation of the veins of the systemic circulation, congestion of the liver, reduction of the size of the left ventricle, and increased filling of the right ventricle. All this results from a sudden contraction of the pulmonic vascular area. The intravenous injection of 3 cm<sup>3</sup> of ephedrine is lethal within 5 min., causes a marked reduction in the volume of all systemic arteries, great distention of the systemic veins, congestion of the liver, great distention of the pulmonary vessels, and increased volume of the left and right ventricles—particularly the left. This depicts a primary contraction of the large systemic arteries and the sequelae therefrom. The intravenous injection of 1 cm<sup>3</sup> of adrenalin induces a most marked dilatation of the aorta and large arteries, dilatation of the pulmonic vessels, reduced size of the systemic veins, and diminution of blood in the portal

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bradycardiac phenomenon was abolished by the intravenous injection of 0.0002 gm. (gr. 1/30) of atropine sulph. B.P. 160/80 over rt. arm and 160/0 over lt. arm. Hb 9.5 gms, r.b.c. 4.2 mill., phosphatase elevated. ECG normal. Oscillometric readings showed abnormally great amplitude of pulsations over the lt. arm, forearm and thigh. Skin temperature readings on the lt. arm averaged from 0.5-1.7° C (1-3° F) higher than on the rt. Venous pressure rt. forearm 12.0 cm., lt. forearm 15.6 cm. water. Oxygen saturation of venous blood from rt. forearm 34.8%, from lt. forearm 90.2%. Circulation time (cyanide) from antecubital spaces of rt. forearm 15.8 cm., lt. forearm 9.2 cm. Biopsies from different parts of the skeleton were taken and submitted to microscopic study.

**Röntgenograms (B)** Definite enlargement of the heart, computed volume 925 cm<sup>3</sup>. The amplitude of pulsations (as noted during fluoroscopy) was increased but diminished on compression of the lt. axillary and femoral arteries. **(C)** Venogram of lt. arm following rapid injection of 20 cc. of 35% diodrast in 40 cc. saline. Note the enlarged veins. Arterio-venous communications could not be demonstrated with certainty.

**Comment:** An instance of polyostotic fibrous dysplasia with multiple congenital phleboarteriectases. These communications were responsible for the abnormal findings of the circulatory system, including cardiac enlargement. See also the publication of Drs. H. M. Stauffer, R. K. Arbuckle and E. E. Aegerter, *Journ. Bone Joint Surg.*, 1941, 23, 323.

system. These findings are interpreted as the result of contraction of the arterioles which causes the blood to back up into the arteries. The inhalation of one capsule of amylnitrite is followed by a diminution in the size of the left ventricle, an increase of the diameters of the aorta and other systemic arteries, more blood in the portal circulation, and slightly less distention of the systemic veins. Active dilatation of the arterial walls, which allows more blood to collect in the arterial side of the circulatory system, is responsible for these changes.

Interesting findings were shown in anaphylactic shock. Rabbits were sensitized by subcutaneous injections of horse serum. Three weeks later, thorotrast was injected under pressure into one femoral artery, penetrating into the inferior segment of the aorta and into the other femoral artery. A roentgenogram was obtained at this instant and again after the intravenous injection of horse serum had produced the typical fall in blood pressure. The arteries of the lower extremities now revealed a marked constriction.

**VARICOSITIES.** Venography shows the exact size, shape and extension of the varicose vessels. There may be a diffuse dilatation present or the proximal and distal portions of a vessel may be found large, with the central portion normal. The valves may be found to be incompetent. Thus, with the patient standing the vessels fill distal to the point of injection, indicating centrifugal blood flow—the roentgenologic equivalent of the clinical “positive Trendelenburg.” With the leg in the horizontal position the contrast medium shows no change in position, or only a slight degree of forward and backward flow. Increase in abdominal pressure causes a distal flow. Massage or muscular activity of the leg causes the contrast medium to reach the deeper veins whereupon it moves centripetally. Likewise in the erect position, muscular activity stops its distal flow and may even cause it to rise. It follows that complete muscular immobility (relaxation) is necessary in employing sclerosing treatment in order to allow sufficient time for the injected substance to act. It is proper to point out that our ideas about blood flow in varicose veins that are gained from contrast injections are probably not quite correct. Contrast media have a specific gravity that is higher than that of blood, and if the blood current is weak a centripetal movement of the contrast medium will take place, it behaves—to use a simile—like mud in a slowly flowing river. It has been actually demonstrated that a contrast medium of the usual concentration will show in varicosities and for the standing position a centrifugal movement, but after reinjection of a two-third dilution of the medium it is immediately noted to flow centripetally. Venography further shows that varicosities do not extend into the deeper tissues of the leg, but it is not unusual to demonstrate that the area and degree of the varicosities are greater than the clinical examination permits one to assume. It has been shown in those instances where the obliterative procedure did not have a lasting effect that refilling and/or canalization occurred through communication with varicose collaterals. Venographic demonstration of the very poor vascularization in the surroundings of callous ulcers explained the poor healing tendency of these ulcers.

**VENOUS OBSTRUCTION AND THROMBOSIS.** *Upper extremity.* A constriction of the axillary vein may occur only when the arm is dependent. Patency of

the vein may be restored when the arm is elevated. The position of the arm, therefore, deserves consideration before a final opinion is rendered as to the presence of obstruction. True thrombosis of the axillary vein is chiefly due to extension of malignancy from the chest or axilla or in conjunction with tuberculosis of lymph nodes. The so-called primary axillary vein thrombosis (the term "intermittent venous occlusion" is used in this connection) occurs in otherwise healthy individuals following local trauma and prolonged unaccustomed effort. Stretching, tears in the wall with repair by intimal hyperplasia, and secondary spasm occur with resulting thickening of the wall. Roentgenograms reveal marked narrowing and irregularity of the lumen. Functional improvement usually results due to the formation of a collateral circulation. In venography of the upper extremity it will be remembered that the basilic vein ordinarily carries the bulk of blood and that the cephalic vein serves as a collateral blood vessel. But if the basilic-axillary system is obstructed, the cephalic vein becomes the chief course for the return of blood and carries it into the subclavian vein. *Lower extremity.* Venography may shed light in instances of obscure foot and leg troubles by detecting evidence of thrombosis that might not have been considered as a causative factor. Reference is made to patients who make a protracted recovery from trauma in the region of the ankle joint with discomfort continuing in spite of all the therapeutic measures tried. Other patients complain of heaviness and pain in the legs, especially after exertion, and develop edema toward the end of the day. This sometimes follows a period of confinement to bed. When old and fairly extensive thrombosis is present, newly formed full-sized veins are noted in the muscles and subcutis. They reveal a rather straight course and their lack of tortuosity distinguishes them from varicosities. This distinction is of importance since confusion with varices has led to sclerosing injections with embolism following Roentgenographic studies in instances of fresh thrombosis have demonstrated a fact that was already known from a few pathological dissection studies, namely, that the process does not start in the large pelvic veins nor in the femoral vein, but rather in the posterior and anterior tibial and peroneal veins, and that from there the thrombosis may propagate in the direction of the blood current until a thrombus sways in the blood stream and gives hardly any signs or symptoms.

*Superior vena cava.* The most common causes of obstruction are aneurysm of the aorta and malignant tumors. The rarest cause is thrombosis. The development of a collateral circulation depends on whether the site of occlusion is above or below the azygos vein. If it is below, all the blood from the upper part of the body returns to the heart through the inf vena cava. Roentgenograms taken following an injection of contrast medium into an antecubital vein show enlarged venous circulatory connections on the homolateral side, with the lateral thoracic vein communicating with intercostal veins and the abdominal epigastric vein. Enlarged intercostal veins apparently may cause shallow scalloping of the ribs like that caused by the arteries in the presence of coarctation of the aorta. If the axillary vein is also found to be occluded, this excludes the possibility that an encircling band at the base of the sup vena cava was the cause of obstruction, but indicates instead

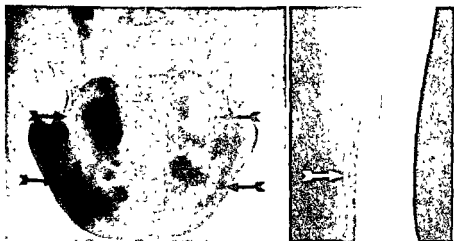
that the thrombosis of the sup. vena cava extended into its main tributaries.

**PRE- AND POSTOPERATIVE VENOUS FLOW.** The following interesting study has been made. 3 to 4 cm<sup>3</sup>. of per-abrodil were injected into the saphenous vein of patients in the recumbent position under fluoroscopic control. The emptying time was determined before and following operations on the stomach, kidneys, bladder and prostate, all of which are known to involve danger of thrombosis and embolism. A pronounced postoperative retardation of the venous blood flow was found, sometimes so much that a standstill was observed. This retardation sets in on the second day after operation and tends to continue as long as the patient stays in bed. A retrograde flow from the saphenous into the femoral vein was observed in several instances. CO<sub>2</sub> inhalation accelerated the venous current much more than active, deep respiration. No or slight retardation was observed in operations not involving danger of embolism, i.e., breast or thyroid surgery. That decreased diaphragmatic activity may play a rôle is shown to be probable by the following experiments on dogs. Bismuthoil or lipiodol was injected into the femoral vein and the time determined which it took the embolus to disappear in the right heart. The average time was 20 seconds. Section of one or both phrenic nerves prolonged the average disappearance time to 28-30 seconds.

Venography is strictly contraindicated in the presence of acute or subacute thrombophlebitis.

**CALCIFICATION OF THE PERIPHERAL ARTERIES** appears chiefly in arteriosclerotics. This process principally affects the peripheral arteries of the muscular type. It has no relation to the central atherosclerotic type, though it happens that this process with its focal-nodular fatty degeneration, ulceration and calcification of the intima, creeps from the abdominal aorta into the femoral arteries and even beyond. The arteriosclerotic calcification gives the typical roentgenographic appearance of fine, granular, beaded deposits and ring-like buckles. Double contours of vessels are outlined for longer or shorter segments. The atherosclerotic deposits are quite irregular, nodular and patchy. The roentgen diagnosis of calcification does not explain vascular disturbances which are more likely to be caused by changes in the intima, though calcification may occur simultaneously. There is usually less calcification present where the vessels cross the larger joints. The lower extremity is much more often affected and often exclusively so. The axillary artery is never and the brachial artery infrequently calcified. The femoral artery is most commonly affected, especially in its middle third, and next in frequency is the posterior tibial artery. Sometimes only deep seated vessels are affected—interossei, radialis, peronei, tibialis. The male sex is much more often affected. The extension and degree of calcification increase definitely with age. Diabetes probably does not cause this process but at least accelerates it, so that calcification appears 10-15 years earlier than in control groups. It can even be observed in diabetic children. It has been shown statistically that the duration of diabetes plays an important rôle. With diabetes lasting more than 10 years, calcification will be detected in more than 90%. Calcification may occur in arteries occluded by the process of thrombo-angiitis, but its incidence here is much lower than in control groups.

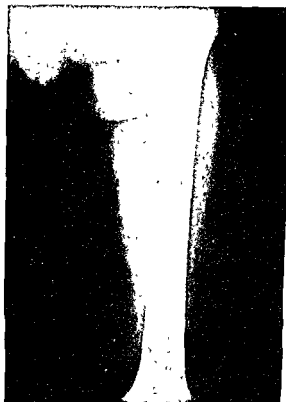
Calcification of arteries may occur in conditions in which there is a disturbance of calcium metabolism. Some of these conditions have manifestations of this disturbed calcium metabolism in the blood serum, which may show an increase or a decrease; in other conditions the blood serum reveals a normal calcium level. The following conditions show an increase in blood serum calcium: 1. Hyperparathyroidism, which may be due to an adenoma or primary hyperplasia of the parathyroid glands. Deposits of lime salts are likewise found in the kidneys or in the para-articular regions. 2. Parathyroid poisoning; this is chiefly concerned with animal experimentation. 3. Hypervitaminosis D. Marked calcification of the media has been produced experimentally in cats and rabbits. Only three human cases have been reported in which this etiology seemed probable. 4. Neoplastic disease



Figs 333, 334—41 yrs, M Intermittent claudication and formation of bleb on big toe for three winters Glycosuria one year ago At present no glycosuria Blood sugar 117 and 134 mgm. Glucose tolerance test reveals mild diabetes Diabetic retinitis and pseudotubercles *Ant view*: extreme degree of calcification in pelvic arteries (bl arrows) *Lat view*: extreme degree of calcification in arterial vessels of leg (wh arrow)

of bone, including multiple myeloma; here the serum calcium may be elevated and generally is if arterial calcification is present due to this etiologic factor. The following conditions show a decrease in blood serum calcium: 1. Chronic nephritis of long duration with... have belonged to the so-called renal rickets. 2. ... in the serum calcium is noted. 3. ... interstitialis, often combined with scleroderma. The roentgenologic diagnosis of vascular calcification is difficult on account of the diffuse lime salt deposits in tendons, fasciae, etc. 2. Osteosclerosis fragilis generalisata (marble disease). 3. Osteogenesis imperfecta. 4. Osteitis deformans (Paget's disease). 5. Hypothyroidism in infants. In one case medication with thyroid extract led to the disappearance of the calcific deposits.

CALCIFICATION OF THE PERIPHERAL VEINS occasionally occurs together with



FIGS 335, 336, 337—37 yrs. History: The patient had nocturia for the past 2 yrs, and more recently felt tired and low weight Findings. The patient was pale, there was no evidence of heart failure The lt. ventricle was moderately hypertrophied, and a faint systolic apical murmur was heard The B P varied between 135/92 and 180/100. The urine showed 150 mgm % of albumin, the urea clearance was 10% The blood showed hb 53 gms rbc 1.8 mill, urea n 84 mgm %, creatinin 5.2 mgm %, plasma CO<sub>2</sub> combining power 25 vol % The Wassermann test was negative Fundi Retinal arteriolosclerosis, grade I

*Roentgenograms* General calcification of the vessels in the thigh region is noted The skull shows thickening and decalcification

*Course.* Following two blood transfusions, hb 8 gms, rbc 2.5 mill, calcium 11.1 mgm %, phosphorus 6.9 mgm %, protein 6 grams with normal a/g ratio ECG Lt axis dev, Q-T interval 0.34 sec, normal (upper limit of normal for a cycle length of



0.77 sec being 0.388 sec). Two mos later the pt went into uremic coma, Chvostek sign

0.333 sec.) Death two days later

*Postmortem:* The heart weighed 310 gms, the coronary arteries showed some degree of atherosclerosis The free edge of the aorta showed calcification The splenic and renal arteries weighed 25 and 50 gms The microscopic diagnosis was congenital hypoplasia of

arterial calcification in any of the previously mentioned conditions; extensive calcification is rare, and probably limited to the superficial veins, a plexiform or dendritic picture may be present. More commonly round, concentric, sharply defined areas of calcification are found, pinpoint to pea-size and with irregular distribution. These are phleboliths and they are found in varicose veins and in subcutaneous and intramuscular hemangiomas.

**CALCIFICATION OF PARASITES.** When calcification of or in peripheral vessels is under consideration, the possibility of calcification in parasites, although rare, should be remembered in differential diagnosis. Cysticerci occasionally undergo calcification and then appear as oval or spindle shaped bodies with pointed ends and averaging 0.2-0.4 by 1.5 cm in size. They are characteristically arranged in a longitudinal fashion, parallel to muscle fibers. Trichinae are uniformly round and appear as multiple pin points. *Filaria Bancrofti* gives rise to oblong or spindle shaped shadows from 0.4-1.5 cm. in length and are located subcutaneously. *Oncocerci* produce round shadows the size of peas, and *Sarcosporidia* show spindle shaped nodules of various sizes in the muscles. Soft tissue roentgenographic technique is recommended. The use of intensifying screens, unless new, should be avoided.

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the kidneys with arteriolonephrosclerosis and interstitial nephritis with vascular and interstitial calcium deposits One parathyroid measured 2 by 3 cm in diameter, and the microscopic diagnosis was adenoma. The skull was thick and decalcified

**Comment** An instance of metastatic vascular calcification associated with bony decalcification in the presence of long standing renal insufficiency. Congenital hypoplasia of the kidneys probably was the primary lesion although chronic glomerulonephritis is the more common cause Hyperparathyroidism was present, whether primary or secondary remained undecided Chronic acidosis, increase in blood phosphorus and increased loss of calcium through the bowels lead to a terminal hypocalcemia This in turn caused characteristic clinical and electrocardiographic findings The calcifying process affected chiefly the medium sized and smaller arterial vessels



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